EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	4	(chondroosteomodulin or TIG2 or GORI-28) and (meder.in. or wendland.in. or john.in. or richter.in. or meyer.in. or forssmann.in.)	US-PGPUB; USPAT	OR	OFF	2007/09/26 17:45
L2	2	chondroosteomodulin	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/09/26 17:46
L3	2	GORI-28 same receptor	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2007/09/26 17:46
L4	1	3 not 2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2007/09/26 17:46
L5	35	tig2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2007/09/26 17:46

Welcome to DIALOG

Dialog level 05.19.02D
? b 411;set files biotech
    26sep07 16:37:10 User219511 Session D703.2
        $0.00   0.115 DialUnits File410
    $0.00 Estimated cost File410
    $0.02 TELNET
    $0.02 Estimated cost this search
    $0.56 Estimated total session cost 0.269 DialUnits
File 411:DIALINDEX(R)

DIALINDEX(R)
  (c) 2007 Dialog

*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
  You have 25 files in your file list.
  (To see banners, use SHOW FILES command)
? s chondroosteomodulin

Your SELECT statement is:
  s chondroosteomodulin

        Items   File
        -----   ----
          1   399: CA SEARCH(R)_1967-2007/UD=14714

  1 file has one or more items; file list includes 25 files.

? save temp; b 399;exs
Temp SearchSave "TD474071840" stored
    26sep07 16:37:32 User219511 Session D703.3
        $0.86   0.291 DialUnits File411
    $0.86 Estimated cost File411
    $0.26 TELNET
    $1.12 Estimated cost this search
    $1.68 Estimated total session cost 0.560 DialUnits

File 399:CA SEARCH(R) 1967-2007/UD=14714
    (c) 2007 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.

    Set Items Description
    --- ----- -----------
Executing TD474071840
  S1   1 CHONDROOSTEOMODULIN
? t s1/7/1

1/7/1
DIALOG(R)File 399:CA SEARCH(R)
(c) 2007 American Chemical Society. All rts. reserv.

  140400709 CA: 140(25)400709w PATENT
  Chondroosteomodulin purified from hemofiltrates for use in the treatment
  or diagnosis of bone or cartilage diseases, obesity, inflammation, and
  skin diseases
  INVENTOR(AUTHOR): Meder, Wolfgang; Wendland, Martin; John, Harald;
  Richter, Rudolf; Meyer, Markus; Forssmann, Wolf-Georg
  LOCATION: Germany,
  ASSIGNEE: IPF Pharmaceuticals GmbH
  PATENT: PCT International ; WO 200439978 A2 DATE: 20040513
  APPLICATION: WO 2003EP11799 (20031024) *DE 10251205 (20021031)
  PAGES: 24 pp. CODEN: PIXXD2 LANGUAGE: German
  PATENT CLASSIFICATIONS:
    CLASS: C12N-015/12A; C07K-014/47B; C07K-016/18B; G01N-033/50B;
  G01N-033/53B; A61K-038/17B; A61P-003/04B; A61P-003/10B; A61P-005/18B;
  A61P-013/12B; A61P-017/00B; A61P-019/00B; A61P-029/00B; A61P-037/00B
    DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ;

CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; EG; ES; FI; GB; GD; GE;
GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LS; LT;
LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NI; NO; NZ; OM; PG; PH; PL; PT; RO;
RU; SC; SD; SE; SG; SK; SL; SY; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VC;
VN; YU; ZA; ZM; ZW; AM; AZ; BY; KG; KZ; MD DESIGNATED REGIONAL: GH; GM; KE
; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK;
EE; ES; FI; FR; GB; GR; HU; IE; IT; LU; MC; NL; PT; RO; SE; SI; SK; TR; BF;
BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG
  SECTION:
    CA202010 Mammalian Hormones
    CA201XXX Pharmacology
    CA213XXX Mammalian Biochemistry
    CA214XXX Mammalian Pathological Biochemistry
    IDENTIFIERS: chondroosteomodulin GORI28 receptor bone cartilage disease
      therapy, sequence chondroosteomodulin human cDNA
    DESCRIPTORS:
  Peptides,biological studies...
      as chondroosteomodulin mimetics, screening for; chondroosteomodulin
      purified from hemofiltrates for use in treatment or diagnosis of bone
      or cartilage diseases, obesity, inflammation, and skin disease
  Cell migration... Chemotaxis...
      chondroosteomodulin in therapeutic modulation of; chondroosteomodulin
      purified from hemofiltrates for use in treatment or diagnosis of bone
      or cartilage diseases, obesity, inflammation, and skin disea
  Chemotherapy...
      chondroosteomodulin modulation of cell migration in support of;
      chondroosteomodulin purified from hemofiltrates for use in treatment or
      diagnosis of bone or cartilage diseases, obesity, inflammation,
  Human...
      chondroosteomodulin purified from hemofiltrates for use in treatment or
      diagnosis of bone or cartilage diseases, obesity, inflammation, and
      skin diseases
  Growth factors,animal...
      chondroosteomodulin; chondroosteomodulin purified from hemofiltrates
      for use in treatment or diagnosis of bone or cartilage diseases,
      obesity, inflammation, and skin diseases
  Bone,disease...
      degenerative, treatment of; chondroosteomodulin purified from
      hemofiltrates for use in treatment or diagnosis of bone or cartilage
      diseases, obesity, inflammation, and skin diseases
  Immunoassay...
      diagnostic, for blood chondroosteomodulin; chondroosteomodulin purified
      from hemofiltrates for use in treatment or diagnosis of bone or
      cartilage diseases, obesity, inflammation, and skin diseases
  Immunity...
      disorder, treatment of; chondroosteomodulin purified from hemofiltrates
      for use in treatment or diagnosis of bone or cartilage diseases,
      obesity, inflammation, and skin diseases
  Antirheumatic agents... Antiarthritics... Antiobesity agents...
  Antidiabetic agents...
      effectors of chondroosteomodulin as; chondroosteomodulin purified from
      hemofiltrates for use in treatment or diagnosis of bone or cartilage
      diseases, obesity, inflammation, and skin diseases
  Kidney,disease...
      failure, treatment of; chondroosteomodulin purified from hemofiltrates
      for use in treatment or diagnosis of bone or cartilage diseases,
      obesity, inflammation, and skin diseases
  Primers(nucleic acid)...
      for detection of components of GOR-28/chondroosteomodulin system;
      chondroosteomodulin purified from hemofiltrates for use in treatment or
      diagnosis of bone or cartilage diseases, obesity, inflammation
  Bone,disease...
      fracture, treatment of; chondroosteomodulin purified from hemofiltrates
      for use in treatment or diagnosis of bone or cartilage diseases,
      obesity, inflammation, and skin diseases
  G protein-coupled receptors...
      GORI-28, endogenous ligand for; chondroosteomodulin purified from
      hemofiltrates for use in treatment or diagnosis of bone or cartilage
      diseases, obesity, inflammation, and skin diseases
  Electrolytes,biological...

imbalances of, treatment of; chondroosteomodulin purified from
hemofiltrates for use in treatment or diagnosis of bone or cartilage
diseases, obesity, inflammation, and skin diseases
Diagnosis...
immunodiagnosis, blood chondroosteomodulin as indicator in;
chondroosteomodulin purified from hemofiltrates for use in treatment or
diagnosis of bone or cartilage diseases, obesity, inflammation, and
Diagnosis...
mol., blood chondroosteomodulin as indicator in; chondroosteomodulin
purified from hemofiltrates for use in treatment or diagnosis of bone
or cartilage diseases, obesity, inflammation, and skin diseas
Diabetes mellitus...
non-insulin-dependent, treatment of; chondroosteomodulin purified from
hemofiltrates for use in treatment or diagnosis of bone or cartilage
diseases, obesity, inflammation, and skin diseases
Peptide library...
screening of, for chondroosteomodulin mimetics; chondroosteomodulin
purified from hemofiltrates for use in treatment or diagnosis of bone
or cartilage diseases, obesity, inflammation, and skin disease
Phosphates,biological studies...
secretion of, treatment of; chondroosteomodulin purified from
hemofiltrates for use in treatment or diagnosis of bone or cartilage
diseases, obesity, inflammation, and skin diseases
Parathyroid gland,disease... Hypoparathyroidism... Osteoporosis...
Cartilage,disease... Connective tissue,disease... Rheumatic diseases...
Arthritis... Obesity... Kidney,disease...
treatment of; chondroosteomodulin purified from hemofiltrates for use
in treatment or diagnosis of bone or cartilage diseases, obesity,
inflammation, and skin diseases
CAS REGISTRY NUMBERS:
688069-27-4P amino acid sequence; chondroosteomodulin purified from
hemofiltrates for use in treatment or diagnosis of bone or cartilage
diseases, obesity, inflammation, and skin diseases
7440-70-2 biological studies, secretion of, treatment of;
chondroosteomodulin purified from hemofiltrates for use in treatment or
diagnosis of bone or cartilage diseases, obesity, inflammation, and
skin diseases
207788-36-1 184384-94-9 186637-50-3 184384-93-8 chondroosteomodulin
purified from hemofiltrates for use in treatment or diagnosis of bone
or cartilage diseases, obesity, inflammation, and skin diseases
688069-28-5 688069-29-6 688069-30-9 688069-31-0 688069-32-1
688069-33-2 688069-34-3 688069-35-4 688069-36-5 688069-37-6
688069-38-7 688069-39-8 688069-40-1 primer for detection of
components of GOR-28/chondroosteomodulin system; chondroosteomodulin
purified from hemofiltrates for use in treatment or diagnosis of bone
or cartilage diseases, obesity, inflammation, and skin diseases
688069-49-0 unclaimed nucleotide sequence; chondroosteomodulin purified
from hemofiltrates for use in the treatment or diagnosis of bone or
cartilage diseases, obesity, inflammation, and skin diseases
? b 411;set files biotech
26sep07 16:37:54 User219511 Session D703.4
$4.77 0.380 DialUnits File399
$2.75 1 Type(s) in Format 7
$2.75 1 Types
$7.52 Estimated cost File399
$0.26 TELNET
$7.78 Estimated cost this search
$9.46 Estimated total session cost 0.940 DialUnits
File 411:DIALINDEX(R)

DIALINDEX(R)
(c) 2007 Dialog

*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
You have 25 files in your file list.
(To see banners, use SHOW FILES command)
? s TIG2

Your SELECT statement is:

s TIG2

Items File
----- ----
10   5: Biosis Previews(R)_1926-2007/Sep W4
 1   8: Ei Compendex(R)_1884-2007/Sep W3
 3   24: CSA Life Sciences Abstracts_1966-2007/Jun
11   34:
 1   45: EMCare_2007/Sep W3
 6   71: ELSEVIER BIOBASE_1994-2007/Aug W4
 8   73: EMBASE_1974-2007/Sep 27
 4 135: NewsRx Weekly Reports_1995-2007/Aug W4
 3 144: Pascal_1973-2007/Sep W3
10 155: MEDLINE(R)_1950-2007/Sep 25
 1 266: FEDRIP_2007/Sep
 1 315: ChemEng & Biotec Abs_1970-2007/Aug
 2 357: Derwent Biotech Res.__1982-2007/Sep W1
 7 399: CA SEARCH(R)_1967-2007/UD=14714

14 files have one or more items; file list includes 25 files.

? save temp; b 155,5,8,24,34,45,71,73,135,144,266,315,357,399;exs;rd
Temp SearchSave "TB474072343" stored
26sep07 16:38:30 User219511 Session D703.5
$0.85 0.289 DialUnits File411
$0.85 Estimated cost File411
$0.26 TELNET
$1.11 Estimated cost this search
$10.57 Estimated total session cost 1.229 DialUnits

SYSTEM:OS - DIALOG OneSearch
File 155:MEDLINE(R) 1950-2007/Sep 25
(c) format only 2007 Dialog
File 5:Biosis Previews(R) 1926-2007/Sep W4
(c) 2007 The Thomson Corporation
File 8:Ei Compendex(R) 1884-2007/Sep W3
(c) 2007 Elsevier Eng. Info. Inc.
File 24:CSA Life Sciences Abstracts 1966-2007/Jun
(c) 2007 CSA.
File 34:
File 45:EMCare 2007/Sep W3
(c) 2007 Elsevier B.V.
File 71:ELSEVIER BIOBASE 1994-2007/Aug W4
(c) 2007 Elsevier B.V.
File 73:EMBASE 1974-2007/Sep 27
(c) 2007 Elsevier B.V.
File 135:NewsRx Weekly Reports 1995-2007/Aug W4
(c) 2007 NewsRx
File 144:Pascal 1973-2007/Sep W3
(c) 2007 INIST/CNRS
File 266:FEDRIP 2007/Sep
Comp & dist by NTIS, Intl Copyright All Rights Res
File 315:ChemEng & Biotec Abs 1970-2007/Aug
(c) 2007 DECHEMA
File 357:Derwent Biotech Res. _1982-2007/Sep W1
(c) 2007 The Thomson Corp.
File 399:CA SEARCH(R) 1967-2007/UD=14714
(c) 2007 American Chemical Society
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IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.

Set Items Description
--- ----- -----------
Executing TB474072343
S1   68 TIG2
S2   28 RD (unique items)
? t s2/7/1-28;bye

2/7/1   (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

24342714 PMID: 17635925
Chemerin, a novel adipokine that regulates adipogenesis and adipocyte metabolism.

Goralski Kerry B; McCarthy Tanya C; Hanniman Elyisha A; Zabel Brian A; Butcher Eugene C; Parlee Sebastian D; Muruganandan Shanmugam; Sinal Christopher J
Department of Pharmacology and College of Pharmacy, Dalhousie University, Halifax, Nova Scotia B3H 1X5, Canada.

Obesity is an alarming primary health problem and is an independent risk factor for type II diabetes, cardiovascular diseases, and hypertension. Although the pathologic mechanisms linking obesity with these co-morbidities are most likely multifactorial, increasing evidence indicates that altered secretion of adipose-derived signaling molecules (adipokines; e.g. adiponectin, leptin, and tumor necrosis factor alpha) and local inflammatory responses are contributing factors. Chemerin (RARRES2 or %%%TIG2%%%) is a recently discovered chemoattractant protein that serves as a ligand for the G protein-coupled receptor CMKLR1 (ChemR23 or DEZ) and has a role in adaptive and innate immunity. Here we show an unexpected, high level expression of chemerin and its cognate receptor CMKLR1 in mouse and human adipocytes. Cultured 3T3-L1 adipocytes secrete chemerin protein, which triggers CMKLR1 signaling in adipocytes and other cell types and stimulates chemotaxis of CMKLR1-expressing cells. Adenoviral small hairpin RNA targeted knockdown of chemerin or CMKLR1 expression impairs differentiation of 3T3-L1 cells into adipocytes, reduces the expression of adipocyte genes involved in glucose and lipid homeostasis, and alters metabolic functions in mature adipocytes. We conclude that chemerin is a novel adipose-derived signaling molecule that regulates adipogenesis and adipocyte metabolism.
Record Date Created: 20070914
Date of Electronic Publication: 20070716

2/7/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

22486188 PMID: 16904155
Characterization of the human chemerin receptor--ChemR23/CMKLR1--as co-receptor for human and simian immunodeficiency virus infection, and identification of virus-binding receptor domains.

Martensson Ulrika E A; Fenyo Eva Maria; Olde Bjorn; Owman Christer
Division of Molecular Neurobiology, Wallenberg Neuroscience Center, Lund University, SE-223 62, Sweden. Ulrika.Martensson@med.lu.se

Studies were conducted to elucidate co-receptor spectrum and function of the inflammatory receptor, CMKLR1/ChemR23, which was recently identified as the receptor for the cystatin-like chemoattractant, %%%TIG2%%%, also named chemerin. An infection model was applied based on stably transfected NP-2.CD4 host cells expressing various co-receptor constructs and exposed to panels of HIV-1, HIV-2 and SIV primary isolates. In a panel of 27 HIV-1 isolates tested, 12 isolates could use CMKLR1/ChemR23. As expected from a relatively high sequence homology with the extracellular domains of CCR3, HIV-1 isolates showing R3 tropism were particularly efficient in using CMKLR1/ChemR23. In addition, 5 out of 7 HIV-2 isolates and 13 out of 15 SIV (SMM-3 origin) used CMKLR1/ChemR23, in accordance with the previously

documented ability of these isolates to use several co-receptors. In order to define important extracellular epitopes for the viral interaction, a hybrid receptor model was applied. This was based on the fact that the rat receptor, although structurally very similar to the human orthologue, was inefficient as viral co-receptor. When the rat receptor was "humanized" to include regions unique to the human receptor (N-terminus or second extracellular loop), exposure to HIV-1, HIV-2 and SIV isolates resulted in infection. The relative importance of the two critical receptor regions differed between HIV-1/HIV-2 on the one hand and SIV on the other. The results strongly support that the chemerin receptor, in the presence of CD4, functions as a "minor co-receptor" promoting infection by these classes of viruses.
Record Date Created: 20061024
Record Date Completed: 20061214
Date of Electronic Publication: 20060810

2/7/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15568908 PMID: 15974952
Discovery of novel regulatory peptides by reverse pharmacology: spotlight on chemerin and the RF-amide peptides metastin and QRFP.

Kutzleb Christian; Busmann Annette; Wendland Martin; Maronde Erik
IPF PharmaCeuticals GmbH, Feodor-Lynen-Str. 31, D-30625 Hannover, Germany. c.kutzleb@ipf-pharmaceuticals.de

Reverse pharmacology is a screening technology that matches G protein-coupled receptors (GPCRs) with unknown cognate ligands in cell-based screening assays by detection of agonist-induced signaling pathways. One decade spent pursuing orphan GPCR screening by this technique assigned over 30 ligand/receptor pairs and revealed previously known or novel undescribed ligands, mostly of a peptidic nature. In this review, we describe the discovery, characterization of the structural composition, biological function, physiological role and therapeutic potential of three recently identified peptidic ligands. These are metastin, QRFP in a context of five RF-amide genes described in humans and the chemoattractant, chemerin. Metastin was initially characterized as a metastasis inhibitor. Investigations using ligand/receptor pairing revealed that metastin was involved in a variety of physiological processes, including endocrine function during pregnancy and gonad development. The novel RF-amide QRFP is implicated in food intake and aldosterone release from the adrenal cortex in the rat. Chemerin, first described as %%%TIG2%%%, is upregulated in tazarotene-treated psoriatic skin. By GPCR screening, bioactive chemerin was isolated from ovarial carcinoma fluid as well as hemofiltrate. Characterization as a chemoattractant for immature dendritic cells and analysis of the expression profile of metastin and its receptor suggested a physiological role of chemerin as a mediator of the immune response, inflammatory processes and bone development. (139 Refs.)
Record Date Created: 20050624
Record Date Completed: 20050822

2/7/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15458147 PMID: 15792532
The mouse chemerin receptor gene, mcmklr1, utilizes alternative promoters for transcription and is regulated by all-trans retinoic acid.

Martensson Ulrika E A; Bristulf Jesper; Owman Christer; Olde Bjorn
Division of Molecular Neurobiology, Wallenberg Neuroscience Center, BMC A12, SE-221 84 Lund, Sweden. Ulrika.Martensson@mphy.lu.se

Gene (Netherlands) Apr 25 2005, 350 (1) p65-77, ISSN 0378-1119--
Print Journal Code: 7706761
Publishing Model Print
Document type: Comparative Study; Journal Article; Research Support,
Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
CMKLR1 (chemoattractant-like receptor 1) is a G-protein-coupled receptor
implicated in cartilage and bone development and is expressed in organs
like the parathyroid gland, brain, and lung. The receptor is also expressed
in dendritic cells and in macrophages where it acts as a co-receptor for
entry of HIV/SIV isolates into human CD4(+) cells. Recently, a protein
named "chemerin" (also known as %%%TIG2%%%) was isolated from human
inflammatory fluids and hemofiltrate and found to be the endogenous ligand
for CMKLR1. We have previously described the genomic organization of the
cmklr1 gene and characterized its promoter in mouse neuroblastoma NB4 1A3
cells. In the present study we identify a second transcript, cmklr1b, in
mouse microglia BV2 cells. Cmklr1b is transcribed from an alternative
promoter with a transcription start site located 6780 bp downstream of the
previously identified exon 1 (cmklr1a). The cmklr1b promoter lacks a TATA
box but contains two CCAAT boxes in opposite directions. 5' Deletion
analysis of the promoter region in BV2 cells using a luciferase reporter
gene assay indicates two regions, between 623-755 bp and 56-125 bp upstream
of transcription start site, to be important for promoter function. The
proximal promoter region includes both CCAAT boxes, and site-directed
mutagenesis separately within these elements revealed that only the forward
CCAAT element was important for transcription. Although the forward CCAAT
element is essential for transcription electrophoretic mobility shift and
super-shift assays demonstrated that both CCAAT elements actually bind
nuclear proteins from BV2 cells and identified the binding factor as NFY.
Real-time reverse transcriptase-PCR experiments of cmklr1b expression in
all-trans retinoic acid (ATRA)- stimulated BV2 cells showed strong
up-regulation of receptor transcript. Luciferase reporter gene assay of the
promoter in ATRA-stimulated BV2 cells confirmed that transcriptional
activity of the cmklr1b promoter is increased by ATRA. However, deletion
analysis could not identify an ATRA-responsive element within the promoter
region suggesting that gene activation is likely to occur through
alternative mechanisms. The results emphasise a possible role of cmklr1 in
bone modelling.
Record Date Created: 20050418
Record Date Completed: 20050525

2/7/5   (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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15171722 PMID: 15522723
A three-step purification strategy for isolation of hamster %%%TIG2%%%
from CHO cells: characterization of two processed endogenous forms.
Busmann Annette; Walden Michael; Wendland Martin; Kutzleb Christian;
Forssmann Wolf-Georg; John Harald
IPF PharmaCeuticals GmbH, Feodor-Lynen-Str. 31, D-30625 Hannover,
Germany.
Journal of chromatography. B, Analytical technologies in the biomedical
and life sciences (United States) Nov 25 2004, 811 (2) p217-23, ISSN
1570-0232--Print Journal Code: 101139554
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
We have recently isolated a bioactive, circulating protein of human
tazarotene-induced gene-2 (%%%TIG2%%%) as the natural ligand of the orphan
receptor ChemR23. Here we describe a simplified method for the isolation of
hamster %%%TIG2%%% protein from Chinese hamster ovary (CHO) cell
supernatant. Using a heparin-affinity column followed by two reversed phase
chromatography steps resulted in the isolation of pure biologically active
material. Two processed bioactive forms of Chinese hamster %%%TIG2%%% were

identified by Edman sequencing and matrix-assisted laser
desorption/ionization-time-of-flight mass spectrometry (MALDI-TOF-MS) mass
fingerprint analysis, representing the amino acid residues T20 to F156, and
T20 to A155 of the 163 amino acid propeptide. Comparison with the predicted
aa-sequence indicates a mutation or modification within the C-terminal end
of the peptide.
Record Date Created: 20041103
Record Date Completed: 20050324

2/7/6   (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2007 Dialog. All rts. reserv.

14637786 PMID: 14675762
Characterization of human circulating %%%TIG2%%% as a ligand for the
orphan receptor ChemR23.
Meder W; Wendland M; Busmann A; Kutzleb C; Spodsberg N; John H; Richter R
; Schleuder D; Meyer M; Forssmann W G
IPF PharmaCeuticals GmbH, Feodor-Lynen-Str. 31, D-30625, Hannover,
Germany. c.kutzleb@ipf-pharmaceuticals.de
FEBS letters (Netherlands) Dec 18 2003, 555 (3) p495-9, ISSN
0014-5793--Print Journal Code: 0155157
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The orphan receptor ChemR23 is a G-protein coupled receptor (GPCR) with
homology to neuropeptide and chemoattractant receptors. Tazarotene, a
synthetic retinoid activating retinoic acid receptor (RAR), up-regulates
tazarotene-induced gene-2 (%%%TIG2%%%). The function and molecular target
of this protein are now described. By means of reverse pharmacology
screening using a peptide library generated from human hemofiltrate, we
have isolated and identified %%%TIG2%%% as the natural ligand of ChemR23
and report the specific molecular form of the bioactive, circulating
%%%TIG2%%%, representing the amino-acid residues 21 to 154 of the 163 amino
acid-containing prepropeptide. Based on the expression pattern of ChemR23
and %%%TIG2%%% , the physiological role in bone development, immune and
inflammatory responses and the maintenance of skin is now being
investigated.
Record Date Created: 20031216
Record Date Completed: 20040123

2/7/7   (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2007 Dialog. All rts. reserv.

13632787 PMID: 11840383
Primary IgA nephropathy with low histologic grade and disease
progression: is there a "point of no return"?
Lai Fernand Mac-Moune; Szeto Cheuk Chun; Choi Paul Cheung Lung; Li Philip
Kam Tao; Tang Nelson Leung Sang; Chow Kai Ming; Lui Siu Fai; Wong Teresa
Yuk Hwa; Ho Kelvin K L; To Ka Fai
Department of Anatomical and Cellular Pathology, The Chinese University
of Hong Kong, Prince of Wales Hospital, Hong Kong, China. fmlai@cuhk.edu.hk
American journal of kidney diseases - the official journal of the
National Kidney Foundation (United States) Feb 2002, 39 (2) p401-6,
ISSN 1523-6838--Electronic Journal Code: 8110075
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Histologic low-grade chronic renal lesions in 144 adults with primary
immunoglobulin A (IgA) nephropathy were correlated with clinical parameters
of disease progression over a median follow-up of 93 months. Using
chronicity-based histologic grading, 50, 59, and 35 patients were
glomerular grade (GG) 1a, GG1b, and GG2; 83 and 61 patients were

tubulointerstitial grade (TIG) 1 and %%%TIG2%%% ; and 25 patients had hyaline arteriolosclerosis. On follow-up, GG and TIG were predictive of disease progression by impairment of renal function, development of hypertension, and significant proteinuria (>1 g/d). Hyaline arteriolosclerosis correlated only with the development of hypertension. Histologic lesions GG1a or TIG1 predicted a significant low risk for disease progression compared with other renal lesions, regardless of the renal manifestation at the time of biopsy. Combined GG1a, TIG1, and isolated hematuria at the time of biopsy enhanced the sensitivity to determine early IgA nephropathy and to define a nonearly cohort with a higher risk of disease progression appropriate for recruitment into clinical therapeutic trials within realistic time frames. The significant risk of progression in other low-grade lesions, such as GG1b or %%%TIG2%%%, suggests that the point of no return in IgA nephropathy may occur much earlier than perceived and that delayed biopsy in these patients no longer may be justified. Copyright 2002 by the National Kidney Foundation, Inc.
  Record Date Created: 20020212
  Record Date Completed: 20020304

2/7/8   (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12966324 PMID: 11109537
  Establishment and characteristics of four sub-strains of F344 rats reared on various low protein and low energy diets.
  Shumiya S; Kuramoto K; Itoh H; Kaneko M
  Department of Laboratory Animal Science, Tokyo Metropolitan Institute of Gerontology, Japan.
  Experimental animals / Japanese Association for Laboratory Animal Science (JAPAN) Jul 2000, 49 (3) p153-61, ISSN 1341-1357--Print
Journal Code: 9604830
  Publishing Model Print
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed
  Four sub-strains, reared by sib-mating and having for their origin the F344/DuCrj strain of rats, were established by feeding with different levels of low protein and low energy diets, and their characteristics investigated. The amounts of crude protein (CP) and digestible energy (DE) in the four diets were 17.6%-3.0 kcal, 10.5%-2.5 kcal, 8.4%-2.0 kcal, and 10.5%-2.5 kcal, respectively, and the four sub-strains established here were provisionally designated as F344/Tig1, F344/%%%Tig2%%%, F344/Tig3 and F344/Tig4, respectively. Intakes of nitrogen-corrected metabolizable energy (MEn) did not differ, and a large intake of digestible crude protein (DCP) was observed in F344/Tig1 rats. The body weight of rats provided with lower-nutrient diets showed a tendency to decrease until the F2 generation, but no change among the generations was seen subsequently, and the same compiled differences in protein content were maintained. Similar transitions were observed in the lifetime rearing test. Though F344/Tig3 rats, which were reared on minimum nutrients, showed a tendency to delayed puberty, we were easily able to breed four pairs in every generation using procedures similar to those used for other strains of rats. There were no differences among the F344/Tig1 to -3 strains of rats in body length, digestive tract length, or organ weight per body weight, and all the rats had a normal range of biochemical values. But the F344/Tig4 showed a high glutamic-oxaloacetic transaminase (GOT), and a tendency to decreased liver function and a shorter lifespan. These sub-strains of F344 rats clarified differences in fatty acid compositions, such as docosahexaenoic acid (DHA) in serum, liver and the brain. The rats were intended to be useful animal models for the study of nutritional environments and their influence on the memory and learning.
  Record Date Created: 20001211
  Record Date Completed: 20001228

2/7/9   (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12498344 PMID: 10440928
  1,25 dihydroxyvitamin D3 and dexamethasone induce the cyclooxygenase 1 gene in osteoclast-supporting stromal cells.
  Adams A E; Abu-Amer Y; Chappel J; Stueckle S; Ross F P; Teitelbaum S L; Suva L J
  Division of Bone and Mineral Metabolism, Beth Israel-Deaconess Medical Center and Harvard Medical School, Boston, Massachusetts 02215, USA. aadams@bidmc.harvard.edu
  Journal of cellular biochemistry (UNITED STATES) Sep 15 1999, 74 (4) p587-95, ISSN 0730-2312--Print Journal Code: 8205768
  Publishing Model Print
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed
  Commitment of members of the monocyte/macrophage family to the bone resorptive phenotype, in vitro, requires contact, of these osteoclast precursors, with osteoblasts or related stromal cells. The osteoclast-inductive properties of these stromal cells are typically expressed, however, only in the presence of steroid hormones such as 1,25 dihydroxyvitamin D (1,25D3) and dexamethasone (DEX). To gain insight into the means by which steroid treated accessory cells induce osteoclast differentiation we asked, using differential RNA display (DRD), if gene expression by this stromal cell population differs from that of their untreated, non-osteoclastogenic counterpart. We identified four known genes specifically expressed by 1,25D3/DEX-treated ST2 stromal cells: 1) a family of rat organic anion transporters, 2) Na/K ATPase ss-subunit, 3) tazarotene-induced gene 2 (%%%TIG2%%%), and 4) prostaglandin G/H synthase I, or cyclooxygenase 1 (Cox-1). The regulation of these genes in 1,25D3/DEX-treated ST2 cells was demonstrated by Northern blot analysis of treated (osteoclast-supporting) and untreated (non-osteoclast-supporting) ST2 cells; the genes have a limited and specific tissue mRNA expression pattern. Northern blot analysis of treated and untreated ST2 cell total RNA using either a DRD-derived Cox-1 cDNA or a Cox-1 specific oligonucleotide confirmed the steroid regulation of Cox-1 mRNA. Surprisingly, there is no detectable expression by untreated or steroid exposed ST2 cells, of Cox-2, the classical regulated cyclooxygenase isoform. In contrast to 1, 25D3/DEX, serum treatment rapidly induces Cox-2 mRNA, substantiating the capacity of ST2 cells to express the gene. These data establish that steroid induction of the osteoclastogenic properties of stromal cells is attended by Cox gene expression, a phenomenon consistent with the capacity of eicosinoids to impact the resorptive process. The response of osteoclast-supporting ST2 cells to 1,25D3/DEX treatment may be one prostaglandin-mediated event which specifically involves Cox-1 regulation. Copyright 1999 Wiley-Liss, Inc.
  Record Date Created: 19991012
  Record Date Completed: 19991012

2/7/10   (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11385477 PMID: 9204961
  Tazarotene-induced gene 2 (%%%TIG2%%%), a novel retinoid-responsive gene in skin.
  Nagpal S; Patel S; Jacobe H; DiSepio D; Ghosn C; Malhotra M; Teng M; Duvic M; Chandraratna R A
  Department of Biology, Allergan, Inc., Irvine, California 92713, U.S.A.
  Journal of investigative dermatology (UNITED STATES) Jul 1997, 109 (1) p91-5, ISSN 0022-202X--Print Journal Code: 0426720
  Contract/Grant No.: AR39915; AR; NIAMS
  Publishing Model Print
  Document type: Journal Article; Research Support, U.S. Gov't, P.H.S.
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed
  Retinoids exert their biologic effects through two families of nuclear receptors, retinoic acid receptors (RARs) and retinoid X receptors (RXRs),

which belong to the superfamily of steroid/thyroid hormone nuclear receptors. By using a subtraction hybridization approach, we have identified a cDNA sequence %%%TIG2%%% (Tazarotene-induced gene 2), whose expression is up-regulated by the treatment of skin raft cultures by an RAR beta/gamma-selective anti-psoriatic synthetic retinoid tazarotene [AGN 190168/ethyl 6-[2-(4,4-dimethylthiochroman-6-yl)-ethynyl] nicotinate]. The retinoid-mediated up-regulation in the expression of %%%TIG2%%% was confirmed by Northern blot analysis. Upon sequencing, %%%TIG2%%% was found to be a cDNA whose complete sequence was not in the GenBank and EMBL data bases. The %%%TIG2%%% cDNA is 830 bp long and encodes a putative protein product of 164 amino acids. %%%TIG2%%% is neither expressed nor induced by tazarotene in primary keratinocyte and fibroblast cultures. Thus, %%%TIG2%%% is expressed and induced by tazarotene only when keratinocytes and fibroblasts form a tissue-like 3-dimensional structure. We further demonstrate that RAR-specific retinoids increase %%%TIG2%%% mRNA levels. In contrast, neither RXR-specific retinoids nor 1,25-dihydroxyvitamin D3 increased %%%TIG2%%% levels. Finally, we demonstrate that %%%TIG2%%% is expressed at high levels in nonlesional psoriatic skin but at lower levels in the psoriatic lesion and that its expression is up-regulated in psoriatic lesions after topical application of tazarotene.
  Record Date Created: 19970717
  Record Date Completed: 19970717

13858429 BIOSIS NO.: 199799492489
Vascular endothelial growth factor (VEGF) in tumours predicts recurrence
  and progression in T1 bladder cancer
AUTHOR: Crew Jeremy; O'Brien Tim; Fuggle Sue; Bicknell Roy; Cranston David;
  Harris Adrian L
AUTHOR ADDRESS: Oxford, UK**UK
JOURNAL: Journal of Urology 157 (4 SUPPL.): p51 1997 1997
CONFERENCE/MEETING: 92nd Annual Meeting of the American Urological
Association New Orleans, Louisiana, USA April 12-17, 1997; 19970412
ISSN: 0022-5347
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

13336403 BIOSIS NO.: 199698804236
TIG1 and %%%TIG2%%% (Tazarotene Induced Genes 1 and 2) are novel retinoic
  acid receptor-responsive genes in skin
AUTHOR: Nagpal Sunil (Reprint); Patel Sheetal; Asano Arisa T; Johnson Alan;
  Duvic Madeleine; Chandraratna Roshantha A S
AUTHOR ADDRESS: Dep. Biol., Allergan Inc., Irvine, CA, USA**USA
JOURNAL: Journal of Investigative Dermatology 106 (4): p818 1996 1996
CONFERENCE/MEETING: Annual Meeting of the Society for Investigative
Dermatology Washington, D.C., USA May 1-5, 1996; 19960501
ISSN: 0022-202X
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

0002802396   IP ACCESSION NO: 6683822
Characterization of human circulating %%%TIG2%%% as a ligand for the orphan
receptor ChemR23

Imhof, Beat
IPF PharmaCeuticals GmbH, Feodor-Lynen-Str. 31, D-30625, Hannover, Germany

ABSTRACT:
  The orphan receptor ChemR23 is a G-protein coupled receptor (GPCR) with
homology to neuropeptide and chemoattractant receptors. Tazarotene, a
synthetic retinoid activating retinoic acid receptor (RAR), up-regulates
tazarotene-induced gene-2 (%%%TIG2%%%). The function and molecular target
of this protein are now described. By means of reverse pharmacology
screening using a peptide library generated from human hemofiltrate, we
have isolated and identified %%%TIG2%%% as the natural ligand of ChemR23
and report the specific molecular form of the bioactive, circulating
%%%TIG2%%%, representing the amino-acid residues 21 to 154 of the 163 amino
acid-containing prepropeptide. Based on the expression pattern of ChemR23
and %%%TIG2%%%, the physiological role in bone development, immune and
inflammatory responses and the maintenance of skin is now being
investigated.

11735181 Genuine Article#: 689BG Number of References: 35
Title: Accuracy of the ImmunoCyt (TM) assay in the diagnosis of
  transitional cell carcinoma of the urinary bladder
Author(s): Feil G (REPRINT) ; Zumbragel A; Paulgen-Nelde HJ; Hennenlotter J
  ; Maurer S; Krause S; Bichler KH; Stenzl A
Corporate Source: Univ Tubingen,Dept Urol,Hoppe Seyler Str 3/D-72076
  Tubingen//Germany/ (REPRINT); Univ Tubingen,Dept Urol,D-72076
  Tubingen//Germany/
Journal: ANTICANCER RESEARCH, 2003, V23, N2A (MAR-APR), P963-967
ISSN: 0250-7005 Publication date: 20030300
Publisher: INT INST ANTICANCER RESEARCH, EDITORIAL OFFICE 1ST KM
  KAPANDRITIOU-KALAMOU RD KAPANDRITI, PO BOX 22, ATHENS 19014, GREECE
Language: English Document Type: ARTICLE
Abstract: The ImmunoCyt(TM) assay (Diagnocure Inc.Quebec, Canada) is a new
  immunocytological fluorescence test for identifying two different
  mucins and a high-molecular-weight glycosylated carcinoembryonic
  antigen (CEA) present in tumours originating from transitional
  epithelial cells. The test promises a higher diagnostic sensitivity in
  transitional cell carcinoma (TCC) of the bladder than voided urine
  cytology. Our study was designed to evaluate this test especially for
  TaG1 carcinomas, which are characterised by a low detection rate in
  urinary cytology. A total of 121 spontaneous urine samples of 92
  patients (age range 28 to 86, mean 62.5 years) were examined. The
  samples were taken from patients suspected of having TCC (41 out of
  121) or tumor recurrence (46 out of 121), or who were part of a
  follow-up protocol (34 out of 121). Cystoscopy was practised in all
  patients. The ImmunoCyt(TM) test was carried out according to the
  manufacturer's protocol. For cytology cytospins were made from the same
  urine samples and stained according to the method of Papanicolaou. One
  hundred and thirteen specimens could be evaluated. In 87 cystoscopy
  and/or histology were negative. There was histological evidence of 7
  pTaG1, 4 pTaG2,. 8 pT1G2/G3 and 7 pT2G2/G3 TCC. As for ImmunoCyt(TM)
  and cytology, specificity was 83.9% and 91.9%, respectively. A
  combination of either test indicated 81.6% specificity,. The
  sensitivity amounted to 38.5% and 34.6%, respectively:. and the

combined sensitivity to 53.8%. The sensitivity, for TaG1 carcinomas was
14.3% each, for TaG2 carcinomas 25% and 50%, for %%%TIG2%%%/G3
carcinomas it amounted to 37.5% each, while for T2G2/G3 carcinomas it
was 71.4% and 42.9%, respectively. The higher sensitivity of the
ImmunoCyt(TM) test as compared to urinary cytology renders improved
identification of exfoliated tumour cells in bladder cancer possible.
In our study, however, the expected increase . it detecting TaG1
carcinomas was not found. Because of its lower specificity, the test
should only be used in combination with voided urine cytology. On
account of its low sensitivity, the ImmunoCyt(TM) test cannot replace
cystoscopy (with biopsy) in the diagnosis and monitoring of bladder
cancer.

2/7/15   (Item 2 from file: 34)
DIALOG(R)File 34:

04722199 Genuine Article#: UC787 Number of References: 0
Title: TIG1 AND %%%TIG2%%% (TAZAROTENE INDUCED GENE-1 AND GENE-2) ARE NOVEL
    RETINOIC ACID RECEPTOR-RESPONSIVE GENES IN SKIN
Author(s): NAGPAL S; PATEL S; ASANO AT; JOHNSON A; DUVIC M; CHANDRARATNA
    RAS
Corporate Source: ALLERGAN PHARMACEUT INC,DEPT BIOL/IRVINE//CA/92715;
    ALLERGAN PHARMACEUT INC,DEPT CHEM/IRVINE//CA/92715; UNIV TEXAS,SCH
    MED,DEPT DERMATOL/HOUSTON//TX/00000; UNIV TEXAS,SCH MED,DEPT
    MED/HOUSTON//TX/77030
Journal: JOURNAL OF INVESTIGATIVE DERMATOLOGY, 1996, V106, N4 (APR), P78
ISSN: 0022-202X
Language: ENGLISH Document Type: MEETING ABSTRACT

2/7/16   (Item 1 from file: 45)
DIALOG(R)File 45:EMCare
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01065039   EMCare No: 34174265
 Primary IgA nephropathy with low histologic grade and disease
progression: Is there a "point of no return"?
 Mac-Moune Lai F.; Szeto C.C.; Choi P.C.L.; Li P.K.T.; Tang N.L.S.; Chow
K.M.; Lui S.F.; Wong T.Y.H.; Ho K.K.L.; To K.F.
 F. Mac-Moune Lai, Department of Anatomical Pathology, Prince of Wales
Hospital, Shatin Hong Kong
 AUTHOR EMAIL: fmlai@cuhk.edu.hk
 American Journal of Kidney Diseases (AM. J. KIDNEY DIS.) (United States
) 2002, 39/2 (401-406)
 CODEN: AJKDD ISSN: 0272-6386
 DOCUMENT TYPE: Journal ; Article
 LANGUAGE: ENGLISH   SUMMARY LANGUAGE: ENGLISH
 NUMBER OF REFERENCES: 17
 RECORD TYPE: Abstract

 Histologic low-grade chronic renal lesions in 144 adults with primary
immunoglobulin A (IgA) nephropathy were correlated with clinical parameters
of disease progression over a median follow-up of 93 months. Using
chronicity-based histologic grading, 50, 59, and 35 patients were
glomerular grade (GG) 1a, GG1b, and GG2; 83 and 61 patients were
tubulointerstitial grade (TIG) 1 and %%%TIG2%%%; and 25 patients had
hyaline arteriolosclerosis. On follow-up, GG and TIG were predictive of
disease progression by impairment of renal function, development of
hypertension, and significant proteinuria (>1 g/d). Hyaline
arteriolosclerosis correlated only with the development of hypertension.
Histologic lesions GG1a or TIG1 predicted a significant low risk for
disease progression compared with other renal lesions, regardless of the
renal manifestation at the time of biopsy. Combined GG1a, TIG1, and
isolated hematuria at the time of biopsy enhanced the sensitivity to
determine early IgA nephropathy and to define a nonearly cohort with a
higher risk of disease progression appropriate for recruitment into
clinical therapeutic trials within realistic time frames. The significant
risk of progression in other low-grade lesions, such as GG1b or %%%TIG2%%%,
suggests that the point of no return in IgA nephropathy may occur much

earlier than perceived and that delayed biopsy in these patients no longer·
may be justified. (c) 2002 by the National Kidney Foundation, Inc.
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2/7/17   (Item 1 from file: 135)
DIALOG(R)File 135:NewsRx Weekly Reports
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0000457128   (THIS IS THE FULLTEXT)
Researchers from Sweden, Switzerland and the United States report recent
findings in virology
Virus Weekly, February 27, 2007, p.165

DOCUMENT TYPE: Expanded Reporting LANGUAGE: English
RECORD TYPE: FULLTEXT
AUDIENCE: Professional
WORD COUNT: 1106

TEXT: Virology research advances have been reported from Sweden,
Switzerland and the United States.
    Study 1: Fresh data on virology are presented in the report
"Characterization of the human chemerin receptor--ChemR23/CMKLR1--as
co-receptor for human and simian immunodeficiency virus infection, and
identification of virus-binding receptor domains." According to a study
from Sweden, "Studies were conducted to elucidate co-receptor spectrum and
function of the inflammatory receptor, CMKLR1/ChemR23, which was recently
identified as the receptor for the cystatin-like chemoattractant,
%%%TIG2%%%, also named chemerin. An infection model was applied based on
stably transfected NP-2.CD4 host cells expressing various co-receptor
constructs and exposed to panels of HIV-1, HIV-2 and SIV primary
isolates.".
    "In a panel of 27 HIV-1 isolates tested, 12 isolates could use
CMKLR1/ChemR23. As expected from a relatively high sequence homology with
the extracellular domains of CCR3, HIV-1 isolates showing R3 tropism were
particularly efficient in using CMKLR1/ChemR23. In addition, 5 out of 7
HIV-2 isolates and 13 out of 15 SIV (SMM-3 origin) used CMKLR1/ChemR23, in
accordance with the previously documented ability of these isolates to use
several co-receptors. In order to define important extracellular epitopes
for the viral interaction, a hybrid receptor model was applied. This was
based on the fact that the rat receptor, although structurally very similar
to the human orthologue, was inefficient as viral co-receptor. When the rat
receptor was "humanized" to include regions unique to the human receptor
(N-terminus or second extracellular loop), exposure to HIV-1, HIV-2 and SIV
isolates resulted in infection. The relative importance of the two critical
receptor regions differed between HIV-1/HIV-2 on the one hand and SIV on
the other," wrote U.E. Mertensson and colleagues, Lund University.
    The researchers concluded: "The results strongly support that the
chemerin receptor, in the presence of CD4, functions as a "minor
co-receptor" promoting infection by these classes of viruses."
    Mertensson and colleagues published their study in(Characterization of
the human chemerin receptor--ChemR23/CMKLR1--as co-receptor for human and
simian immunodeficiency virus infection, and identification of
virus-binding receptor domains. Virology, 2006;355(1):6-17).
    For more information, contact U.E. Mertensson, Wallenberg Neuroscience
Center, Division of Molecular Neurobiology, Lund University, SE-223 62,
Sweden.
    Study 2: Interferon defense system evasion model may be key in
infection success.
    Researchers in Switzerland explained, "Bovine viral diarrhea virus
(BVDV), together with Classical swine fever virus (CSFV) and Border disease
virus (BDV) of sheep, belongs to the genus of the . BVDV is either
cytopathic (cp) or noncytopathic (ncp), as defined by its effect on
·cultured cells."
    M. Schweizer and colleagues, University of Bern, reported that
"[i]nfection of pregnant animals with the ncp biotype may lead to the birth
of persistently infected calves that are immunotolerant to the infecting
viral strain. In addition to evading the adaptive immune system, BVDV
evades key mechanisms of innate immunity.
   · "Previously," the authors continued, "we showed that ncp BVDV inhibits

the induction of apoptosis and alpha/beta interferon (IFN-alpha/beta) synthesis by double-stranded RNA (dsRNA). Here, we report that

(i) both ncp and cp BVDV block the induction by dsRNA of the Mx protein (which can also be induced in the absence of IFN signaling);

(ii) neither biotype blocks the activity of IFN; and

(iii) once infection is established, BVDV is largely resistant to the activity of IFN-alpha/beta but

(iv) does not interfere with the establishment of an antiviral state induced by IFN-alpha/beta against unrelated viruses."

"The results of our study suggest that, in persistent infection, BVDV is able to evade a central element of innate immunity directed against itself without generally compromising its activity against unrelated viruses ('nonself') that may replicate in cells infected with ncp BVDV," Schweizer and coworkers found. "This highly selective 'self' and 'nonself' model of evasion of the interferon defense system may be a key element in the success of persistent infection in addition to immunotolerance initiated by the early time point of fetal infection."

Schweizer and colleagues published their study in the ('Self' and 'nonself' manipulation of interferon defense during persistent infection: Bovine viral diarrhea virus resists alpha/beta interferon without blocking antiviral activity against unrelated viruses replicating in its host cells. J Virol, 2006;80(14):6926-6935).

For additional information, contact M. Schweizer, University of Bern, Institute Vet. Virology, Laenggass Str 122, CH-3001 Bern, Switzerland.

Study 3: African green monkey species and simian immunodeficiency virus strains must be matched for SIV infection models.

According to recent research published in the , "The simian immunodeficiency viruses (SIV) naturally infect a wide range of African primates, including African green monkeys (AGM). Despite moderate to high levels of plasma viremia in naturally infected AGM, infection is not associated with immunodeficiency."

"We recently reported that SIVagmVer90 isolated from a naturally infected vervet AGM induced AIDS following experimental inoculation of pigtailed macaques. The goal of the present study was to evaluate the replication of this isolate in two species of AGM, sabaeus monkeys ( ) and vervets ( ).

"Inoculation of sabaeus AGM with SIVagmVer90 resulted in low and variable primary and set-point viremia (<10 to 10 copies/mL). In contrast," said the authors, "inoculation of vervet AGM with either SIVagmVer90 or blood from a naturally infected vervet (Verl) resulted in high primary viremia and moderate plateau levels, similar to the range seen in naturally infected vervets from this cohort."

"CD4+ T cells remained stable throughout infection, even in AGM with persistent high viremia. Despite the lack of measurable lymphadenopathy," S. Goldstein and colleagues at the U.S. National Institutes of Health wrote, "infection was associated with an increased number of Ki-67 T cells in lymph node biopsies, consistent with an early antiviral immune response."

Investigators concluded, "The preferential replication of SIVagmVer in vervet versus sabaeus AGM shows that it is critical to match AGM species and SIV strains for experimental models of natural SIV infection."

Goldstein and colleagues published their study in the (Comparison of simian immunodeficiency virus SIVagmVer replication and CD4+ T-cell dynamics in vervet and sabaeus African green monkeys. J Virol, 2006;80(10):4868-4877).

For additional information, contact V.M. Hirsch, NIAID, Molecular Microbiology Laboratory, National Institutes of Health, 4 Center Dr., Bldg 4, Rm B1-41, Bethesda, MD 20892, USA.

Keywords: Bethesda, Maryland, United States, Simian Immunodeficiency Virus, African Green Monkey, Plasma Viremia, Experimental Model, Immune Response.

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2/7/18   (Item 2 from file: 135)
DIALOG(R)File 135:NewsRx Weekly Reports
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TEXT: Data published by Lund University, Sweden, researchers advance medical knowledge.

This trend article about Lund University, Sweden, is an immediate alert from NewsRx to identify developing directions of research.

Study 1: Fresh data on virology are presented in the report "Characterization of the human chemerin receptor--ChemR23/CMKLR1--as co-receptor for human and simian immunodeficiency virus infection, and identification of virus-binding receptor domains." According to a study from Sweden, "Studies were conducted to elucidate co-receptor spectrum and function of the inflammatory receptor, CMKLR1/ChemR23, which was recently identified as the receptor for the cystatin-like chemoattractant, %%%TIG2%%%, also named chemerin. An infection model was applied based on stably transfected NP-2.CD4 host cells expressing various co-receptor constructs and exposed to panels of HIV-1, HIV-2 and SIV primary isolates."

"In a panel of 27 HIV-1 isolates tested, 12 isolates could use CMKLR1/ChemR23. As expected from a relatively high sequence homology with the extracellular domains of CCR3, HIV-1 isolates showing R3 tropism were particularly efficient in using CMKLR1/ChemR23. In addition, 5 out of 7 HIV-2 isolates and 13 out of 15 SIV (SMM-3 origin) used CMKLR1/ChemR23, in accordance with the previously documented ability of these isolates to use several co-receptors. In order to define important extracellular epitopes for the viral interaction, a hybrid receptor model was applied. This was based on the fact that the rat receptor, although structurally very similar to the human orthologue, was inefficient as viral co-receptor. When the rat receptor was "humanized" to include regions unique to the human receptor (N-terminus or second extracellular loop), exposure to HIV-1, HIV-2 and SIV isolates resulted in infection. The relative importance of the two critical receptor regions differed between HIV-1/HIV-2 on the one hand and SIV on the other," wrote U.E. Mertensson and colleagues, Lund University.

The researchers concluded: "The results strongly support that the chemerin receptor, in the presence of CD4, functions as a "minor co-receptor" promoting infection by these classes of viruses."

Mertensson and colleagues published their study in(Characterization of the human chemerin receptor--ChemR23/CMKLR1--as co-receptor for human and simian immunodeficiency virus infection, and identification of virus-binding receptor domains. Virology, 2006;355(1):6-17).

For more information, contact U.E. Mertensson, Wallenberg Neuroscience Center, Division of Molecular Neurobiology, Lund University, SE-223 62, Sweden.

Study 2: Intraoperative sentinel node (SN) detection enhances the staging of invasive bladder cancer.

"We evaluated intraoperative SN detection in patients with invasive bladder cancer during radical cystectomy in conjunction with extended lymphadenectomy. A total of 75 patients with invasive bladder cancer underwent radical cystectomy with extended lymphadenectomy. SNs were identified by preoperative lymphoscintigraphy, intraoperative dynamic lymphoscintigraphy and blue dye detection," scientists writing in the report.

"An isotope (70 MBq Tc-99m-nanocolloid) and Patent Blue blue dye were injected peritumorally via a cystoscope. Excised lymph nodes were examined ex vivo using a handheld gamma probe. Identified SNs were evaluated by extended serial sectioning, hematoxylin and eosin staining, and immunohistochemistry. At lymphadenectomy an average of 40 nodes (range 8 to 67) were removed," said F. Liedberg and colleagues, Lund University.

"Of 75 patients 32 (43%) were lymph node positive, of whom 13 (41%) had all lymph node metastases located only outside of the obturator spaces. An SN was identified in 65 of 75 patients (87%). In 7 patients an SN was recognized when the nodal basins were assessed with the gamma probe after lymphadenectomy and cystectomy. Of the 32 lymph node positive cases 26 (81%) had a positive (metastatic) SN. Thus, the false-negative rate was 6

of 32 cases (19%)."

According to study data, "five false-negative cases had macrometastases and/or perivesical metastases. In 9 patients (14%) the SN contained micrometastases (less than 2 mm), in 5 of whom the micrometastasis was the only metastatic deposit. SN detection is feasible in invasive bladder cancer, although the false-negative rate was 19% in this study."

"Extended serial sectioning and immunohistochemistry revealed micrometastases in SNs in 9 patients and radio guided surgery after the completion of lymphadenectomy identified SNs in an additional 7. We believe that the technique that we used in this study improved nodal staging in these 16 of 65 patients (25%)," scientists concluded.

Liedberg and colleagues published their study in the (Intraoperative sentinel node detection improves nodal staging in invasive bladder cancer. J Urol, 2006;175(1):84-88).

Additional information can be obtained by contacting F. Liedberg, Lund University, Dept. Urology, SE-22185 Lund, Sweden.

Study 3: Investigators describe methods for tick prevention in a population living in a highly endemic area in a recent issue of the .

Researchers in Sweden conducted a study "to describe environmental and personal tick-preventive measures and their predictors, taken by a population living in a highly tick-endemic area."

Louise Stjernberg and Johan Berglund at Lund University explained, "Owing to the recent confirmation of human tick-borne encephalitis cases, vaccination against tick-borne encephalitis was offered to the population living in the endemic area through the use of leaflets and media campaigns. At the time of the initial dose, information and enrollment to this cohort study was carried out. Participants' characteristics, frequency of tick-bites and preventive measures were included in questionnaires. Logistic analysis was used to determine behavioral differences in activities taken in order to prevent tick bites."

"In total, 70% of the permanent residents had themselves vaccinated before the next tick season. Of the participants studied, 356/517 (69%) regularly took preventive measures in their environment and/or personally. Women in particular, and those previously treated for a tick-borne disease, took significantly more preventive measures," the researchers reported.

"When analyzing all variables together, spending less time in a tick-endemic area and being tick-bitten during the latest tick season significantly increased the probability of taking preventive measures," stated Stjernberg and Berglund. "After being tick-bitten, men were more inclined to start taking preventive measures than women. Awareness of the risks caused by living in a high tick-endemic area influenced the participant's daily life through preventive activities."

"Public health action should be considered, thus encouraging out-of-door activities for the population without anxiety as to the risks of contracting tick-borne disease after being tick-bitten," the authors advised.

Stjernberg and Berglund published their study in the (Tick prevention in a population living in a highly endemic area. Scand J Public Health, 2005;33(6):432-438).

For additional information, contact Louise Stjernberg, Blekinge Institute of Technology, School of Health Science, S-37179 Karlskrona, Sweden. louise.stjernberg@bth.se.

Keywords: Karlskrona, Sweden, Tick-Borne Encephalitis, Tick-Borne Disease, Vector-Borne Disease, Public Health, , Vectors and Zoonoses.

TEXT: Virology is the focus of recent research from Sweden, the United Kingdom and the United States.

Study 1: Fresh data on virology are presented in the report "Characterization of the human chemerin receptor--ChemR23/CMKLR1--as co-receptor for human and simian immunodeficiency virus infection, and identification of virus-binding receptor domains." According to a study from Sweden, "Studies were conducted to elucidate co-receptor spectrum and function of the inflammatory receptor, CMKLR1/ChemR23, which was recently identified as the receptor for the cystatin-like chemoattractant, %%%TIG2%%%, also named chemerin. An infection model was applied based on stably transfected NP-2.CD4 host cells expressing various co-receptor constructs and exposed to panels of HIV-1, HIV-2 and SIV primary isolates.".

"In a panel of 27 HIV-1 isolates tested, 12 isolates could use CMKLR1/ChemR23. As expected from a relatively high sequence homology with the extracellular domains of CCR3, HIV-1 isolates showing R3 tropism were particularly efficient in using CMKLR1/ChemR23. In addition, 5 out of 7 HIV-2 isolates and 13 out of 15 SIV (SMM-3 origin) used CMKLR1/ChemR23, in accordance with the previously documented ability of these isolates to use several co-receptors. In order to define important extracellular epitopes for the viral interaction, a hybrid receptor model was applied. This was based on the fact that the rat receptor, although structurally very similar to the human orthologue, was inefficient as viral co-receptor. When the rat receptor was "humanized" to include regions unique to the human receptor (N-terminus or second extracellular loop), exposure to HIV-1, HIV-2 and SIV isolates resulted in infection. The relative importance of the two critical receptor regions differed between HIV-1/HIV-2 on the one hand and SIV on the other," wrote U.E. Mertensson and colleagues, Lund University.

The researchers concluded: "The results strongly support that the chemerin receptor, in the presence of CD4, functions as a "minor co-receptor" promoting infection by these classes of viruses."

Mertensson and colleagues published their study in(Characterization of the human chemerin receptor--ChemR23/CMKLR1--as co-receptor for human and simian immunodeficiency virus infection, and identification of virus-binding receptor domains. Virology, 2006;355(1):6-17).

For more information, contact U.E. Mertensson, Wallenberg Neuroscience Center, Division of Molecular Neurobiology, Lund University, SE-223 62, Sweden.

Study 2: Over 7 years of combination antiretroviral therapy (CART), risk of initial virological failure of treatment has been cut at least in half, according to a multicohort analysis (1996-2002).

According to a study from England, "Triple-combination antiretroviral therapy for human immunodeficiency virus infection has been in use for almost a decade, but the extent to which treatment success has changed is uncertain. We examined risk of initial virological failure of CART according to the year of starting therapy. We included subjects from five complete clinic cohorts in Europe and Canada who started CART without previous antiretroviral therapy from 1996-2002 with one or more pre-CART viral load (VL) measurement and CD4 count."

"Based on the first VL measurement from 6-12 months after CART initiation, virological failure was defined as a VL of more than 500 copies/mL. We used the following 3 inclusion strategies:

"1) including all subjects, with missing VL measurement counted as virological failure (n=3825; strategy A);

"2) including all subjects with VL measurement (n=3120; strategy B); and

"3) including all subjects receiving antiretroviral therapy at VL measurement (n=2890; strategy C)," wrote F.C. Lampe and colleagues, Royal Free & University College Medical School, London.

"From 1996-2002, risk of virological failure fell from 38.9% to 24.8% for strategy A, 28.4% to 12.0% for strategy B, and 22.8% to 8.2% for strategy C," the researchers continued. "Estimated relative reductions in risk (95% confidence interval) over the 7-year period, adjusted for cohort, demographic factors, pre-CART VL and CD4 count, and previous AIDS, were 48% (39-56%), 64% (53-73%), and 79% (69-85%) for strategies A, B, and C,

respectively. Reductions in risk were greatest from 1996-1999, with weaker trends subsequently. Trends remained but were attenuated after further adjustment for the starting regimen.

Over a 7-year period of CART use in clinical practice, risk of initial virological failure of treatment has halved at least, the researchers concluded. "These data suggest the trend is due to improvements in CART regimens and greater effectiveness of their use."

Lampe and colleagues published their study in (Changes over time in risk of initial virological failure of combination antiretroviral therapy - A multicohort analysis, 1996 to 2002. Arch Intern Med, 2006;166(5):521-528).

For more information, contact F.C. Lampe, Royal Free & University College, School of Medicine, Dept. Primary Care & Population Science, Royal Free Campus, Rowland Hill St., London NW3 2PF, England.

Study 3: Oral oseltamivir and A-322278 effectively reduces influenza viral replication.

"We developed an immunocompromised murine model of influenza virus infection and demonstrated comparable efficacy of oral oseltamivir and A-322278 (both given at dosages of 10 mg/kg/day) in reducing viral replication, decreasing weight loss, and prolonging survival.

"Once the treatment was discontinued, severe combined immunodeficient (SCID) mice had progressive viral replication and clinical decline," scientists writing in the report.

According to M.G. Ison and colleagues at the University of Virginia, "Drug-resistant variants were detected in 4 (29%) of 14 and 2 (13%) of 15 mice (both BALB/c and SCID) treated with oseltamivir or A-322278, respectively; no resistant variants were detected in placebo-treated mice.

"Amino acid substitutions in the hemagglutinin receptor-binding site at aa 137 or 225 were detected in cloned resistant isolates. A substitution in the neuraminidase (NA) active site (Arg292Lys) was detected in the cloned virus recovered from an oseltamivir-treated mouse."

"This model would be useful for elucidation of the molecular mechanisms of resistance to NA inhibitors and for testing of anti-influenza therapy options that might prevent the emergence of resistant variants," the authors concluded.

Ison and colleagues published their study in the (Comparative activities of oseltamivir and A-322278 in immunocompetent and immunocompromised murine models of influenza virus infection. J Infect Dis, 2006;193(6):765-772).

Additional information can be obtained by contacting L.V. Gubareva, University of Virginia, Dept. Internal Medicine, POB 800473, Charlottesville, VA 22908, USA.

Keywords: Charlottesville, Virginia, United States, Influenza Virus, Viral Replication, Oseltamivir, Therapeutic Efficacy, SCID Mice.

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2/7/20   (Item 4 from file: 135)
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0000375580   (THIS IS THE FULLTEXT)
Study results from Lund University in the area of virology published
Virus Weekly, December 5, 2006, p.91

DOCUMENT TYPE: Expanded Reporting LANGUAGE: English
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TEXT: Fresh data on virology are presented in the report "Characterization of the human chemerin receptor--ChemR23/CMKLR1--as co-receptor for human and simian immunodeficiency virus infection, and identification of virus-binding receptor domains." According to a study from Sweden, "Studies were conducted to elucidate co-receptor spectrum and function of the inflammatory receptor, CMKLR1/ChemR23, which was recently identified as the receptor for the cystatin-like chemoattractant, %%%TIG2%%%, also named chemerin. An infection model was applied based on

stably transfected NP-2.CD4 host cells expressing various co-receptor constructs and exposed to panels of HIV-1, HIV-2 and SIV primary isolates.".

"In a panel of 27 HIV-1 isolates tested, 12 isolates could use CMKLR1/ChemR23. As expected from a relatively high sequence homology with the extracellular domains of CCR3, HIV-1 isolates showing R3 tropism were particularly efficient in using CMKLR1/ChemR23. In addition, 5 out of 7 HIV-2 isolates and 13 out of 15 SIV (SMM-3 origin) used CMKLR1/ChemR23, in accordance with the previously documented ability of these isolates to use several co-receptors. In order to define important extracellular epitopes for the viral interaction, a hybrid receptor model was applied. This was based on the fact that the rat receptor, although structurally very similar to the human orthologue, was inefficient as viral co-receptor. When the rat receptor was "humanized" to include regions unique to the human receptor (N-terminus or second extracellular loop), exposure to HIV-1, HIV-2 and SIV isolates resulted in infection. The relative importance of the two critical receptor regions differed between HIV-1/HIV-2 on the one hand and SIV on the other," wrote U.E. Mertensson and colleagues, Lund University.

The researchers concluded: "The results strongly support that the chemerin receptor, in the presence of CD4, functions as a "minor co-receptor" promoting infection by these classes of viruses."

Mertensson and colleagues published their study in Virology (Characterization of the human chemerin receptor--ChemR23/CMKLR1--as co-receptor for human and simian immunodeficiency virus infection, and identification of virus-binding receptor domains. Virology, 2006;355(1):6-17).

For more information, contact U.E. Mertensson, Wallenberg Neuroscience Center, Division of Molecular Neurobiology, Lund University, SE-223 62, Sweden.

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Keywords: Sweden, Viral, Virology, Virus.

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2/7/21   (Item 1 from file: 266)
DIALOG(R)File 266:FEDRIP
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IDENTIFYING NO.: 160840; 0019; 640 AGENCY CODE: VA
Novel Dendritic Cell Chemoattractant and Receptor
PRINCIPAL INVESTIGATOR: Butcher, Eugene C., M.D.
PERFORMING ORG.: Department of Veterans Affairs, Medical Center, Palo Alto, CA
SPONSORING ORG.: Department of Veterans Affairs, Research and Development (15), 810 Vermont Ave. N.W., Washington, D.C. 20420 United States of America
DATES: 20040811
SUMMARY: RECEPTORS, CHEMOKINE; MONOCYTE CHEMOATTRACTANT PROTEINS; LEUKOCYTES; DENDRITIC CELLS; ANTIGENS
OBJECTIVES
Our goal is to define the role(s) of a novel chemotactic factor %%%TIG2%%% (tazarotene-induced gene 2), and it's receptor, CMKLRI (ChemR23), in leukocyte homing and biology. Extensive preliminary data lead us to hypothesize that this chemoattractant/receptor pair has a significant role in the trafficking of subsets of dendritic cells, key antigen processing andpresenting cells that initiate and regulate immune responses in vivo. RESEARCH PLAN AND METHODS To study dendritic cells and leukocytes to define the role of CMKLRI and %%%TIG2%%% in dendritic cell and leukocyte biology. These studies will be performed on human blood leukocytes and on mouse animal models. We will identify the white blood cells that express CMKLRI then investigate the functional reponse of the CMKLRI+ cells by characterizing their migration to the chemoattractant %%%TIG2%%%. We will also examine the expression and migratory response of mouse leukocytes from CMKLRI knockout animals.

CLINICAL RELEVANCE Proposed studies promise to define an important re

gulator of immune responses, and may suggest novel strategies to enhanceimmunity for vaccination, or to suppress immunity for transplantation, autoimmune and allergic diseases.

2/7/22 (Item 1 from file: 315)
DIALOG(R)File 315:ChemEng & Biotec Abs
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025012 CEABA Accession No.: 05-09-004026 DOCUMENT TYPE: Journal
Title: Acid leaching of Ilmenite into synthetic rutile
AUTHOR: Mackey, T.S.
JOURNAL: Ind. Eng. Chem. Prod. Res. Develop., , Page(s): 9-18
PUBLICATION DATE: Mar 1974 (740300) LANGUAGE: English
ABSTRACT: During the past 20 years, there has been a trend to produce pigments via the chloride process, which has traditionally rused rutile assaying over 95 percent %%%tig2%%% as a feed material. The sulfate process, ground ilmenite is digested with strong sulfuric acid, yielding a titanium sulfate solution which is later hydrolyzed and precipitated to form a tio2 pigment and a solid waste consisting mostly of ferrous sulfate septahydrate crystals. Laboratory methods were developed to produce sythetic rutile from various mature ilmenites. The most effective technique was the use of a rotating heated jar, with hydrochloric acid as the leaching agent to remove iron. Synthetic rutile was formed in a relatively short period of time under controlled conditions. The acid-leaching method could solve the possible future shortage of rutile, particularly if the chlorine is recycled and iron recovered. The remov al of various impurities from ilmenite was studied. For example, as iron was leached from the ilmenite, magnesium, which substitutes for iron, was also removed from the crystal structure. The removal of trace elements in the laboratory experiments was quite similar to remove in nature between source rock ilmenite and altered ilmenite.

2/7/23 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.
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0388191 DBR Accession No.: 2006-01687 PATENT
Diagnosing prostate cancer, by identifying sample of prostate tissue as cancerous when expression of a first or second group of RNA transcripts or translation products is lower or higher in the test sample than in the control sample - a method of diagnosis of prostate cancer involving expression profiling of e.g. glutathione-transferase and estrogen receptor
AUTHOR: SHEKAR M; ZHANG Z; CALDWELL M C; CHEN Z; FAN Z; MCNEAL J E; NOLLEY R; STAMEY T A; WARRINGTON J A; PALMA J F
PATENT ASSIGNEE: AFFYMETRIX INC 2005
PATENT NUMBER: US 20050272052 PATENT DATE: 20051208 WPI ACCESSION NO.: 2006-018828 (200602)
PRIORITY APPLIC. NO.: US 975592 APPLIC. DATE: 20041027
NATIONAL APPLIC. NO.: US 975592 APPLIC. DATE: 20041027
LANGUAGE: English
ABSTRACT: DERWENT ABSTRACT: NOVELTY - Diagnosing prostate cancer in a patient comprising identifying a test sample of prostate tissue as cancerous when expression of at least one of the first group of RNA transcripts or translation products is found to be lower in the test sample than in the control sample, and expression of at least one of the second group of transcripts or translation products is found to be higher in the test sample than in the control sample, is new. DETAILED DESCRIPTION - Diagnosing prostate cancer in a patient comprises: (a) comparing level of expression of at least one RNA transcript or its translation product in a test sample of prostate tissue to level of expression of the at least one transcript or translation product in a control sample of prostate tissue, where the test sample of prostate tissue is suspected of being neoplastic and the control sample is nonmalignant prostate tissue, where the at least one RNA transcript or its translation product is selected from a first or a second group of RNA transcripts or translation products; and (b) identifying the test

sample as cancerous when expression of at least one of the first group of RNA transcripts or translation products is found to be lower in the test sample than in the control sample, and expression of at least one of the second group of transcripts or translation products is found to be higher in the test sample than in the control sample. BIOTECHNOLOGY - Preferred Method: Diagnosing prostate cancer in a patient comprises: (a) comparing level of expression of at least one RNA transcript or its translation product in a test sample of prostate tissue to level of expression of the at least one transcript or translation product in a control sample of prostate tissue, where the test sample of prostate tissue is suspected of being neoplastic and the control sample is nonmalignant prostate tissue, where the at least one RNA transcript or its translation product is selected from a first or a second group of RNA transcripts or translation products, where the first group of RNA transcripts comprises of transcripts of genes selected from GST alpha glutathione S-transferase exon 2 (X65727), Glutathione S-transferase IIa subunit 2 (GST) (M16594), trans-glutaminase (HG4020-HT4290), P15-protease inhibitor 5 (maspin) (U04313), L Arg:Gly amidinotransferase (S68805), KIAA0089 (D42047), RTVP-1 protein (X91911), GSTP1 (glutathione S-transferase pi) (M24485), L-arginine:glycine amidinotransferase (X86401), DNA endothelin-A receptor (D11151), Id1 (HG3342-HT3519), bcl-2 (M14745), Protein Phosphatase Inhibitor Homolog (HG3570-HG3773), pS2 protein (X52003), HAOX (aldehyde oxidase) (L11005), glutaredoxin (X76648), CO-029 (M35252), NADP dependent leukotriene b412-hydroxydehydrogenase (D49387), Glucocorticoid receptor alpha (M10901), Glucocorticoid receptor beta (HG4582-HT4987), ZAKI-4 from skin fibroblast (D83407), syndecan (exon 2-5) (Z48199), S-adenosylmethionine decarboxylase (M21154), hevin-like protein (X86693), gas 1 (L13698), pcHDP7 liver dipeptidyl peptidase IV (X60708), adult male liver squalene epoxidase (D78129), cathepsin H (X16832), oestrogen receptor (X03635), Id-related helix-loop-helix protein Id4 (U28368), apM2 GS2374 (D45370), macrophage capping protein (M94345), nucleotide binding protein (L04510), DNA cystatin A (D88422), Decorin (HG3431-HT3616), RACH1 (U35735), %%%TIG2%%% (tazarotene-induced 2) (U77594), gravin (U81607), H19 RNA (M32053), adipsin/complement factor D (M84526), chondroitin sulfate proteoglycan versican V0splice-variant precursor peptide (U16306), ne1-related protein 2 (D83018), IGFBP6 (insulin-like growth factor I) (X57025), cellular retinol-binding protein (MI1433), laminin B1 chain (M61916), DNA primase (subunit 58) (X74331), complement protein component C7 (J03507), neuronal membrane glycoprotein M6b (U45955), TGF-beta3 (transming growth factor-beta3) (X14885), keratonocyte growth factor (M60828), SPARC/osteonectin (J03040), K+ channel beta subunit (L39833), procollagen C-proteinase enhancer protein (PCOLCE) (L33799), GTPase homolog HeLA cell line 833 nt (S82240), alpha-2 macroglobulin (M11313), thrombospondin (X14787), CAPL protein (M80563), prepro-alpha2(I) collagen (Z74616), pigment epithelium-derived factor (U29953), aspartoacylase kidney 1435 nt (S67156), class I alcohol dehydrogenase (ADH1) alpha subunit (M12963), CRBP (retinol binding protein) (X07438), Ovarian cancer down-regulated myosin heavy chain homolog (Doc1) (U53445), Insulin-like Growth factor 2 (HG3543-HT3739), Prostaglandin D2 synthase (M98539), hIRH (intecrin-alpha) (U19495), G9i) protein alpha-subunit (X04828), tryptase-III 3'-end (M33403), lumican (U21128), TIMP-3: C-terminus region (D45917), 3'UTR of unknown protein (Y09836), novel protein with short consensus repeats of six cysteines (U61374), h-SmLIM (smooth muscle LIM protein) (U46006), LACI (lipoprotein-associated coagulation inhibitor) (M59499), phospholamban (M63603), transcriptional activator hSNF2a (D26155), smooth muscle myosin heavy chain (D10667), erm exon 2,3,4,5 (X96381), telomeric repeat binding factor (TRF1), N2A3 (U97105), GBP-2 (guanylate binding protein isom I) (M55542), metalloproteinase inhibitor (M32304), matrilin-2 precursor, 11-HSD11 (beta-hydroxysteroid dehydrogenase) (M76665), CCK (cholecystokinin) (L00354), apM2 GS2374 (D42047), CYP1B1 (dioxin-inducible cytochrome P450) (U03688), lung amiloride sensitive Na+ channel protein (X76180), PCP4 (PEP19) (U52969), NAT1 (anylamine N-acetyltransferase) (X17059), squalene synthase (X69141), Id-2 (helix-loop-helix protein) (M97796), Zn-alpha2-glycoprotein (X59766), Striated muscle contraction regulatory protein (Id2B) (M96843), Glucocorticoid receptor Beta (HG4582-HT4087), HLH 12R1 helix-loop-helix protein (X69111), PSE-binding factor PTF gamma subunit (U44754), cancellous

bone osteoblast GS3955 (D87119), prostatic secretory protein 57 (U22178), K-sa, (Fibroblast Growth Factor Receptor) (M87770), creatine kinase-B (M16364), ornithine aminotransferase (M29927), epsilon-BP (IgE-binding protein) (M57710), ARL3 (GTP binding protein) (U07151), RNase 4 (D37921), MSP (Beta-microsemiprotein) (M34376), phospholipase C (D42108), lipocortin II (D00017), DBI (diazepam binding inhibitor) (M14200), KIAA0367 (AB002365), MAT8 protein (X93036), protein- tyrosin phosphatase (HU-PP-1) (U14603), imogen38 (Z68747), Cystatin A (D88422), Cytokeratine 15 (X07696), P-450 HFLa (Fetal liver cytochrome P-450) (D00408), Fetal brain (239FB) mRNA from the WAGR region (U57911), Caveolin (Z18951), MLCK (myosin light chain kinase) (U48959), cardiac gap junction protein (X52947), lactate dehydrogenase B (Ec 1.1.1.27) (X13794), KIA0003 (D13628), TRPC1 protein (X89066), unknown protein (D28124), K+ channel beta subunit (L39833), COX7A (cytochrome c oxidase subunit VIIa muscle isoforms (M83186), desmin (M63391), HBNF-1 (nerve growth factor) (M57399), hIRH intercrien-alpha (U19495), fibroblast muscle-type tropomyosin (M12125), SLIM1 (skeletal muscle LIM-protein) (U60115), Adipsin/complement factor D (M84526), Epidermalkeratin-50 kDa type Ie (J00124), n-19 RNA (M32053), Keratin type II 58 kD (M21389), neuronal membrane glycoprotein M6B (U45955), GS TM3 (Glutathione transferase M3) (J05459), unknown protein (U61374), Insulin-like growth factor-2 (IG3543-HT373), IGFBP6 (insulin-like growth factor binding protein 6) (M62402), P-cadherin (X63629), alpha-B crystalline (S45630), MaxiK potassium channel beta (U25138), MLC-2 (myosin light chain) (J02854), caveolin 2 (U32114), SOD3 (extracellular superoxide dismutase) (J02947), ERM (X96381), GLUT5 (Glucose transport-like 5) (M55531), pigment epithelium derived factor (U29953), CRBP (retinol binding protein) (X07438), calcyclin (IG2788-HT289), dehydropyrimidinase related protein-3 (D78014), NECDIN related protein (U35139), CAPL protein (M80563), Mig-2 (Z24725), Heat shock protein 28 kDa (Z23090), smooth muscle gamma-actin (D00654), p68 (Y00097), KIAK002 (D13639), G9i) protein-alpha subunit (adelynate cyclase inhibiting GTP-b) (X04828), BPAG1 (Bullous pemphigoid antigen) (M69225), retinol-binding protein (M11433), TGF beta (transming growth factor-beta type III receptor) ((L07594), aspartoacylase (S67156), ERF-2 (X78992), complement protein component C7 (J03507), Mac-2 binding protein (Li3210), vinculin (M33308), phospholamban (M63603), tissue inhibitor of metalloproteinase 3 (U14394), calponin ((D17408), glypican (hepara sulfate proteoglycan (X54232), keratinocyte growth factor (M60828), trophinin (U04811), TRPM-2 protein (M63379), filamin ABP-280 (actin binding protein) ((X53416), collagen VI alpha 2C-terminal globular domain (Xi5882), GBP-2 (guanylate binding protein II) (M55543), CALLA (common acute lymphoblastic leukemia antigen) (J03779), enigma ((L35240), MT-11 (X76717), ALDHI (RNA mitochondrial aldehyde dehydrogenase) (X05409), breast tumor antigen (U24576), non-muscle alpha-actinin (M95178), pur (pur-alpha) (M96684), N2A3 (U97105), 64 kD autoantigen expressed in thyroid and extra-ocular muscle (X54162), GTPase homolog (S82240), arginase type II (U82256), tryptase-III (M33493), CD3 8 (D84276), muscarinic acetylcholine receptor (M35128), NF-H exon 1 (X15306), tenascin-C 7560 bp (X78565), LPP (IIM protein) (U49957), KIA0172 (D79994), MTIG (clone 14 VS metallothionein-IG) (J03910), smoothelin (Z49989), KIP 2 (Cdk-inhibitor p57 KIP1 (U22398), n-chimaerin (X51408), metallothionein from cadmium-treated cells (V00594), collagen VI alpha-1C-terminal globular domain (X15880), soluble carrier family 39 (zinc transporter) (NM014579.1), secretoglobin family 1A member I (uteroglobin) (NM003357.1), serine or cystrein proteinase inhibitor (NM002639.1), SIAT7E (NM030965), nebulin (NM004543.2), proenkephalin (NM006211.1), aminolevulinate delta dehydratase (BC000977.1), hypothetical protein FIJ20513 (NM017855.i), erythrocyte membrane protein band 4.1-like 3 (AI770004), adipose specific 2, unknown protein (BG109855), syndecan 1 (Z48199), keratin 5 (NM000424.1), cytochrome p450 subfamily 1 (NM000104.2), glutathione S-transferase pi (NM000852.2), phosphorylase glycogen (NM002863.1), zinc finger protein 185 (LIM domain) (NM007150.1), single carrier family 16 AA705628), aminoethyltransferase (NM000480), transmembrane 7 superfamily member 2 (AF096304.1), chemokine (C-X-C motif) ligand 13 (NM006419.1), NEL-like 2 (NM006159.1), D component of complement (adipsin) (NM001928.1), EGF-containing fibulin-like EMP-1 (AI826799), retinol binding protein 1 (NM002899.2), fibulin 1 (Z95331), tissue inhibitor of metalloproteinase 3 (NM00362.2), signal transduction protein (NM005864.1), dihydropyrimidinase-like 3 (NM001387.1), WNT inhibitory factor 1 (NM007191.1), signal transduction protein (SH3 containing) (NM005864.1), collagen type IV alpha 6 (AI889941), suppression of tumorigenicity 5 (NM005418.1), and where the second group of RNA transcripts or translation products are being selected from pyrroline 5-carboxylase reductase (M77836), KIAA0230 (D86983), transcription factor ETR10 (M62831), TGF-beta superfamily (AB000584), intestinal trefoil factor (L08044), aldehyde dehydrogenase 6 (U07919), carcinoma associated antigen GA733-2 (M93036), IQGAP2 (RasGAP-related protein) (U51903), Macmarcks (HG1612-HT1612), KIAA0056 (D29954), SOX-4 protein (X70683), hR-PTPu protein tyrosine phosphatase (X58288), EGR2 (early growth response 2) (J04076), DNA-polymerase gamma (U60325), cystathionine beta synthase, alt splice 3 (HG2383-HT4824), CPBP (DNA-binding protein CPBP) (U44975), skeletal muscle C-protein (X66276), HU-K5 (lysophospholipase homolog) (U67963), fibromodulin (U05291), prostatin (L41351), apolipoprotein E (M12529), hEGR1 (early growth response 1) (X52541), DNA polymerase beta (D29013), GOS3 (L49169), ANK-3 (Ankyrin G) (U13616), Gap junction protein (X04325), Hepsin (X07732), CYP1B1 (dioxin-inducible cytochrome P450 (U03688), T-cell receptor Ti rearranged gamma chain V-J-C (M30894), KIAA00167 (D28589), ornithine decarboxylase (M33764), Tob (D38305), 17-beta-hydroxysteroid dehydrogenase (X87176), homeo box c8 protein (M16938), TRAIL (TNF-related apoptosis inducing ligand (U37518), cellular onco-fos (V01512), ESE-1 (epithelial-specific transcription factor) (U73843), prostate-specific membrane antigen, alternatively spliced (S76978), prostate-specific membrane antigen (M99487), T-cell receptor Ti rearranged gamma chain V-J-C region (M30894), OSF-2os (Osteoblast specific factor 2) (D13666), LDL phospholipase A2 (U24577), MAOA (monoamine oxidase A) (M68840), ALCAM (CD6 ligand) (L38608), UDP-GalNAC:polypeptide N-acetylgalactosaminyl transferase (X92689), NB thymosin beta (D82345), FBP1 (Fructose-1,6 biphosphatase), NMB (X76534), cytochrome c-1 (J04444), ionizing radiation conferring protein (U18321), Myoglobin exon 1 (X00371), Memc (U30999), Clone 23587 sequence (U90914), pyrroline 5-carboxylate synthetase (X94453), ADE2H1 (X53793), (SNX) sorting nexin 1 (U53225), IMPDH2 (inosine monophosphate dehydrogenase type II) (L33842), transcription factor E2F-5 (U31556), propionyl CoA carboxylase beta subunit (S67325), 6-pyruvoyl-tetrahydropterin synthase (D17400), ADP/ATP carrier protein (J02683), nucleoside diphosphate kinase Nm23-H2s (HG1153-HT1153), ornithine decarboxylase (M33764), CLCN3 (X78520), c-fos (V01512), PCC (propionyl-CoA carboxylase beta-subunit) (M31169), adenylsuccinate lyase (X65867), Cctg chaperonine (X74801), SIM2 (U80456), liver gap-junction protein (X04325), C-myc (L00058), HLA-DMB (U15085), carcinoma-associated antigen GA733-2 (M93036), homeo box c8 protein (M16938), GST-1 Hs GTP binding protein (X17219), Brain guanine nucleotide binding protein (M17219), spermidine synthase (M34338), NAD-dependent methylene tetrahydrofolate dehydrogenase cyclohydrolase (E.C. 1.5.1.15) (X16396), C8FW phosphoprotein (AJ000480), NBK apoptotic inducer protein (X89986), TK (transketolase) (L12711), MNK1 (AB000409), fatty acid synthase (S80437), tubulin beta (HG4322-HT4592), testican (X73608), Arg protein kinase-binding protein (X95632), DNA polymerase delta (U21090), IP-30 (gamma-interferon-inducible protein) (J03909, Lutheran blood group glycoprotein (X83425), tyrosine phosphatase 1 non-receptor (HG3187-HT3366), metastasis-associated mta-1 (U35113), (RPS6KA2) ribosomal protein S6 kinase 2 (L06797), transcription factor mef2 alt. splice 2 (HG4668-HT5083), basic transcription factor 44 kDa (HG3748-HT4018), soluble guanylate cyclase large subunit (X66534), transcription factor ETR10 (M62831), orphan G-protein-coupled receptor (L06797), MHC Class II W52 (HG3576-HT3779), prostasin (L41351), M6 antigen (X64364), Mrpl7 (X79865), Ly-GDI (GDP-dissociation inhibitor protein) (L20688), KH type splicing regulatory protein KSRP (U94832), Ia-associated invariant gamma-chain (M13560), HLA-DRB1 (MHC class II beta1) (M33600), transcriptional activator hSNF2b (D26156), USF2 (AD000684), SEP protein (X87904), nested protein (M34677), HOXA9 (class I homeoprotein) (U41813), BRG1 (transcriptional activator) (U29175), KIAA0075 (D38550), eIF3 (translational initiation factor) (U78525), KIAA0113 (D30755), HU-K5 (lysophospholipase homolog) (U67963), ADP/ATP translocase (J03592), inducible poly(A)-binding protein (U33818), KIAA0146 (D63480), NET1 (guanine nucleotide regulatory protein) (U02081), KIAA0162 (D79984), v-ets erythroblastosis virus E26 oncogene

like (AI351043), FBJ murine osteosarcoma viral oncogene homolog B (NMO06732.1), ubiquitin D (NMO06398.1), sialyltransferase I (AI743792), RALBP1 associated Eps domain containing 2 (NM004726.1), chemokine (C-C motif) ligand 19 (U88321.1), transient receptor potential cation channel subfamily M member (NM01736.1), B cell activation gen(S59049.1), eukaryotic translation initiation factor 4E binding protein 1 (AB044548.1), lymphocyte antigen 75 (NM002349), alpha-methylacyl-CoA racemase (NMO14324.1), phosphoprotein regulated by mitogenic pathway (NM 025195.1), RALBP1 associated Eps domain containing 2 (NM004726.1), neuropilin (NRP) and tolloid (TLL)-like 2 (NM018092.1), twist homolog (X99268.1), calcium calmodulin-dependent protein kinase 2 (AA181179), tumor associated calcium signal transducer 1 (NM002354.1), UDP-N-acetylglucosamine phosphorylase I (S73498.1), epithelial cell transforming sequence 2 oncogene (NM01898.1), myosin VI (U90236.2), LIM protein (NM006457.1), claudin 8 (AL049977.1), phosphoprotein regulated by mitogenic pathway (NM025195.1), thymosin beta (NM021992.1), TNF (ligand) superfamily (U57059.1), unknown protein (AV715767), activated leukocyte cell adhesion molecule (NM001627.1), chaperonin containing TCP1 (NM001762.1), phosphoribosylaminoimidazole carboxylase (AA902652), protein (NM23A) (NM000269.1); and (b) identifying the test sample as cancerous when expression of at least one of the first group of RNA transcripts or translation products is found to be lower in the test sample than in the control sample, and expression of at least one of the second group of transcripts or translation products is found to be higher in the test sample than in the control sample. Diagnosing prostate cancer in a patient further comprises determining the level of expression of RNA transcripts using an array of nucleic acid molecules. It also comprises comparing the level of expression of at least one RNA transcript in the test sample to the level of expression of the transcript in the control sample. The method further comprises comparing transcripts or translation products of at least 2-30 of the genes of the first group, and comparing transcripts or translation products of at least 2-29 genes of the second group. It further comprises determining the expression level of maspin (U04313) transcript or its translation product. It also comprises determining the expression level of hepsin (X07732) transcript or its translation product. The method further comprises comparing at least 30-40 of the transcripts or translation products in the first group and 20 of the transcripts or translation products in the second group. At least one RNA transcript or its translation product of the first group of RNA transcripts or translation products comprises the transcript of the gene maspin (U04313). Alternatively, at least one RNA transcript or its translation product of the second group of RNA transcripts comprises the transcript of the gene hepsin (X07732). Preferably, the test sample comprises Gleason grade 4/5 prostate carcinoma cells. The non-malignant prostate tissue is benign prostate hyperplasia tissue. The method further comprises identifying the test sample as Gleason grade 4/5 prostate carcinoma. USE - The method is useful for diagnosing prostate cancer in a patient. EXAMPLE - No relevant example given.(40 pages)

AUTHOR: WITTAMER V; COMMUNI D; VANDENBOGAERDE A; DETHEUX M; PARMENTIER M

ABSTRACT: DERWENT ABSTRACT: NOVELTY - Identifying an agent that modulates the function of an orphan G-protein coupled receptor, ChemR23, is new. DETAILED DESCRIPTION - Identifying an agent that modulates the function of an orphan G-protein coupled receptor, ChemR23, comprises contacting a ChemR23 polypeptide with a tazarotene-induced gene 2 (%%%TIG2%%%) polypeptide in the presence and absence of a candidate modulator to permit the binding of the %%%TIG2%%% polypeptide to the ChemR23 polypeptide, and measuring the binding of the ChemR23 to the %%%TIG2%%% polypeptide, where a decrease in the presence of the candidate modulator, identifies the modulator as an agent that modulates the function of ChemR23. The method further comprises contacting a ChemR23 polypeptide with a candidate modulator, measuring a signaling activity of the ChemR23 polypeptide, where a change in the activity in the presence of the candidate modulator relative to the activity in the absence of the candidate modulator identifies it as an agent that modulates the function of ChemR23, and/or comparing the activity measured in the presence of the candidate modulator to the activity measured in a sample in which the ChemR23 polypeptide is contacted with a %%%TIG2%%% polypeptide at its EC50, where the candidate modulator is identified as an agent that modulates function of ChemR23 when the amount of the activity measured in the presence of the candidate modulator is at least 50% of the amount induced by the %%%TIG2%%% polypeptide present at EC50. INDEPENDENT CLAIMS are also included for the following: (1) detecting the presence, in a sample, of an agent that modulates the function of ChemR23 in a sample, comprising contacting a ChemR23 polypeptide with a %%%TIG2%%% polypeptide in the presence and absence of the sample to permit the binding of the %%%TIG2%%% polypeptide to the ChemR23 polypeptide, and measuring the binding of the ChemR23 to the %%%TIG2%%% polypeptide, where a decrease in the presence of the sample, identifies the presence of an agent that modulates the function of ChemR23 in the sample, and /or contacting a ChemR23 polypeptide with a sample, measuring a signaling activity of the ChemR23 polypeptide, where a change in the activity in the presence of the sample relative to the activity in the absence of the sample identifies an agent that modulates the function of ChemR23, and/or comparing the activity measured in a reaction containing ChemR23 and %%%TIG2%%% polypeptides without the sample to the activity measured with a sample in which the ChemR23 polypeptide is contacted with a %%%TIG2%%% polypeptide at its EC50, where the sample is identified as containing an agent that modulates function of ChemR23 when the amount of the activity measured in the presence of the sample is at least 50% of the amount induced by the %%%TIG2%%% polypeptide present at EC50; (2) modulating the activity of a ChemR23 polypeptide in a cell, comprising delivering an agent that modulates the activity of ChemR23 polypeptide to the cell; (3) an agent identified by any of the methods cited above; (4) a composition comprising the agent of (3); (5) a truncated %%%TIG2%%% peptide comprising any of 12 fully defined sequences of 151-162 amino acids, given in the specification; (6) a nucleotide sequence encoding the truncated %%%TIG2%%% peptide, comprising a fully defined sequence of 471 bp, given in the specification; (7) diagnosing a disease or disorder with dysregulation of ChemR23 signaling, comprising contacting a tissue sample with an antibody specific for a ChemR23 and/or %%%TIG2%%% polypeptide, detecting binding of the antibody to the tissue sample, and comparing the binding detected with a standard, where a difference in binding relative to the standard is diagnostic of a disease or disorder with dysregulation of ChemR23, or isolating nucleic acid from a tissue sample, amplifying a ChemR23 or %%%TIG2%%% polynucleotide, using the nucleic acid as a template, and comparing the amount or sequence of amplified ChemR23 or %%%TIG2%%% polynucleotide produced with a standard, where a difference in the amount or sequence of amplified ChemR23 or %%%TIG2%%% polynucleotide relative to the standard is diagnostic of a disease or disorder; (8) a composition comprising an isolated ChemR23 or %%%TIG2%%% polypeptide;(9) an antibody specific for a ChemR23 or %%%TIG2%%% polypeptide; (10) a kit for screening for agents that modulate the signaling activity of ChemR23, comprising an isolated ChemR23 polypeptide, an isolated polynucleotide encoding a ChemR23 polypeptide or, a cell transformed with a polynucleotide encoding a ChemR23 polypeptide, and packaging materials; (11) a kit for the diagnosis of a disease or disorder with dysregulation of ChemR23,

comprising an isolated ChemR23 polypeptide, an isolated polynucleotide encoding a ChemR23 polypeptide or, a cell transformed with a polynucleotide encoding a ChemR23 polypeptide, and packaging materials; and (12) a non-human mammal having a homozygous null mutation in the gene encoding ChemR23, or that is transgenic for a ChemR23 or %%%TIG2%%% polynucleotide. BIOTECHNOLOGY - Preferred Method: The %%%TIG2%%% polypeptide in the method of identifying an agent that modulates ChemR23 is detectably labeled, preferably with a moiety that is a radioisotope, a fluorophore, a quencher of fluorescence, an enzyme, an affinity tag or an epitope tag. The contacting is performed in or on a cell expressing the ChemR23 polypeptide, or on synthetic liposomes, or on virus-induced budding membranes containing a ChemR23 polypeptide. The method is performed using a membrane fraction from cells expressing a ChemR23 polypeptide. The measuring is performed using label displacement, surface plasmon resonance, fluorescence resonance energy transfer, fluorescence quenching or fluorescence polarization. The agent is a peptide, polypeptide, antibody or antigen-binding fragment, a lipid, carbohydrate, nucleic acid or a small organic molecule. The step of measuring a signaling activity comprises detecting a change in the level of a second messenger, or comprises measurement of guanine nucleotide binding or exchange, adenylate cyclase activity, cAMP, protein kinase C activity, phosphatidylinositol breakdown, diacylglycerol, inositol triphosphates, intracellular calcium, arachinoid acid, MAP kinase activity, tyrosine kinase activity or reporter gene expression, and preferably using an aequorin-based assay. The %%%TIG2%%% polypeptide has at least 31% identity or higher, such as 45%, 55%, 65%, 75%, 85%, 95% or 100% to a fully defined sequence of 163 amino acids (S1), and where it binds specifically to and activates a signaling activity of a ChemR23 polypeptide with a fully defined sequence of 376 amino acids (S2), all given in the specification. The %%%TIG2%%% polypeptide further comprises one or more additions, insertions, deletions or substitutions relative to (S2). Alternatively, the %%%TIG2%%% polypeptide is a truncated %%%TIG2%%% polypeptide with any of 12 fully defined sequences of 151-162 amino acids, given in the specification. The polypeptide additionally comprises additional sequences forming a %%%TIG2%%% fusion protein. The additional sequences are glutathione-S-transferase, maltose binding protein, alkaline phosphatase, thioredoxin, green fluorescent protein, histidine tags or epitope tags. The standard in the method of (7) is a fully defined sequence of 1290 bp, given in the specification. The step of comparing the sequence comprises missequencing, where the standard is a fully defined sequence of 588 bp, given in the specification. The comparing of amount or sequence is performed on a microarray. ACTIVITY - Cytostatic; Antiinflammatory; Immunosuppressive; Osteopathic; Antipsoriatic; Dermatological; Antibacterial; Virucide; Antihelmintic; Gynecological. No biological is data given. MECHANISM OF ACTION - G-Protein-Agonist; G-Protein-Antagonist; Gene-Therapy. USE - The agent or composition is useful for the preparation of a medicament for the treatment of a ChemR23- or %%%TIG2%%% -related disease or disorder, such as cancer, tumor metastasis, inflammatory disease, autoimmune disease, inherited or acquired immune deficiencies, osteoporosis, bone healing, bone tissue grafts, graft rejection, psoriasis, eczema, inflammatory infection, trophic diseases of skin, viral, bacterial and parasitic infections, female infertility and ovarian and uterine tumors. The truncated or full-length %%%TIG2%%% polypeptide is useful for the production of a composition of an isolated ChemR23 or %%%TIG2%%% polypeptide, and/or for the production of a kit for screening agents that modulate the signaling of ChemR23, and/or ligand for ChemR23 (all claimed). EXAMPLE - No suitable example is given.(99 pages)

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Methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adipose tissues

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SECTION:
  CA201010 Pharmacology
  CA203XXX Biochemical Genetics
  CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: diagnosis therapy obesity diabetes insulin resistance biomarker gene, antiobesity antidiabetic drug screening gene biomarker modulator, gene expression profile adipose obesity diabetes insulin resistance diagnosis
DESCRIPTORS:
Gene,animal...
  ADPN, for adiponutrin; methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adipose tissues
DNA microarray technology...
  Affymetrix; methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adipose tissues
Gene,animal...
  ALOX5, for arachidonate 5-lipoxygenase; methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adipose tissu
Gene,animal...
  CMA1, for chymase; methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adipose tissues
Biomarkers...
  diagnostic; methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adipose tissues
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  DUSP4, for dual specificity phosphatase; methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adipose tiss
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  ECHDC1 (enoyl CoA hydratase domain contg. 1); methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adipose
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  ECHDC1, ECHDC3, HADHSC genes for; methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adipose tissues
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  ECHDC1, for enoyl CoA hydratase domain contg. 1; methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adip
Proteins...
  ECHDC3 (enoyl CoA hydratase domain contg. 3); methods of diagnosing and treating obesity, diabetes and insulin resistance by modulators of biomarkers identified by gene expression profiling in adipose

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... Human... Gene expression profiles,animal... Drug screening...
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Diagnosis...
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NPR1, for atrial natriuretic peptide receptor; methods of diagnosing
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PLD3, for phospholipase D; methods of diagnosing and treating obesity,
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PTGER2, for prostaglandin receptor type EP2; methods of diagnosing and
treating obesity, diabetes and insulin resistance by modulators of
biomarkers identified by gene expression profiling in adipose
Gene,animal...
PTGER3, for prostaglandin receptor type EP3; methods of diagnosing and
treating obesity, diabetes and insulin resistance by modulators of
biomarkers identified by gene expression profiling in adipose
Gene,animal...
PTGER4, for prostaglandin receptor type EP4; methods of diagnosing and
treating obesity, diabetes and insulin resistance by modulators of
biomarkers identified by gene expression profiling in adipose
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RARRES2, for retinoid acid receptor responsive 2, (Tig2); methods of
diagnosing and treating obesity, diabetes and insulin resistance by
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SCRN2, for peptidase; methods of diagnosing and treating obesity,
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TM7SF2 (transmembrane 7 superfamily member 2); methods of diagnosing
and treating obesity, diabetes and insulin resistance by modulators of
biomarkers identified by gene expression profiling in adipos
Gene,animal...
TM7SF2, for transmembrane 7 superfamily member 2; methods of diagnosing
and treating obesity, diabetes and insulin resistance by modulators of
biomarkers identified by gene expression profiling in adi
Gene,animal...
TNMD, for tenomodulin; methods of diagnosing and treating obesity,
diabetes and insulin resistance by modulators of biomarkers identified
by gene expression profiling in adipose tissues
Prostanoid receptors...
type EP2, gene PTGER2; methods of diagnosing and treating obesity,
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by gene expression profiling in adipose tissues
Prostanoid receptors...
type EP3, gene PTGER3; methods of diagnosing and treating obesity,
diabetes and insulin resistance by modulators of biomarkers identified
by gene expression profiling in adipose tissues
Prostanoid receptors...
type EP4, gene PTGER4; methods of diagnosing and treating obesity,
diabetes and insulin resistance by modulators of biomarkers identified
by gene expression profiling in adipose tissues
CAS REGISTRY NUMBERS:
876071-57-7 876071-58-8 876071-59-9 876071-63-5 876071-65-7
876071-67-9 876071-69-1 876071-70-4 876071-72-6 876071-74-8
876071-76-0 876071-78-2 876071-80-6 876071-82-8 876071-84-0
876072-35-4 876072-37-6 876072-39-8 876072-41-2 876072-43-4
876072-45-6 876072-47-8 876072-49-0 876072-51-4 876072-53-6
876072-55-8 876072-57-0 876072-59-2 876072-60-5 876072-62-7
876072-64-9 876072-66-1 876072-68-3 876072-70-7 876072-72-9
876072-74-1 876072-76-3 876072-78-5 876072-80-9 876072-82-1
876072-84-3 876072-86-5 876072-88-7 876072-90-1 876072-92-3
876072-94-5 876072-96-7 876072-98-9 876072-99-0 876073-01-7
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876073-14-2 876073-15-3 876073-09-5 876073-10-8 876073-11-9
876073-12-0 876073-13-1 876072-33-2 876073-06-2 876073-07-3 amino
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insulin resistance by modulators of biomarkers identified by gene
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9004-10-8 biological studies, resistance, treating; methods of diagnosing
and treating obesity, diabetes and insulin resistance by modulators of
biomarkers identified by gene expression profiling in adipose tissues
712321-22-7 gene ADPN; methods of diagnosing and treating obesity,
diabetes and insulin resistance by modulators of biomarkers identified
by gene expression profiling in adipose tissues

80619-02-9 gene ALOX5; methods of diagnosing and treating obesity,
  diabetes and insulin resistance by modulators of biomarkers identified
  by gene expression profiling in adipose tissues
97501-92-3 gene CMA1; methods of diagnosing and treating obesity, diabetes
  and insulin resistance by modulators of biomarkers identified by gene
  expression profiling in adipose tissues
9028-40-4 gene HADHSC; methods of diagnosing and treating obesity,
  diabetes and insulin resistance by modulators of biomarkers identified
  by gene expression profiling in adipose tissues
9001-87-0 gene PLD3; methods of diagnosing and treating obesity, diabetes
  and insulin resistance by modulators of biomarkers identified by gene
  expression profiling in adipose tissues
635677-02-0 gene RARRES2; methods of diagnosing and treating obesity,
  diabetes and insulin resistance by modulators of biomarkers identified
  by gene expression profiling in adipose tissues
139858-02-9 441569-68-2 263959-93-9 190149-05-4 337891-24-4
  322309-66-0 340335-14-0 459384-52-2 methods of diagnosing and
  treating obesity, diabetes and insulin resistance by modulators of
  biomarkers identified by gene expression profiling in adipose tissues
876071-56-6 876071-60-2 876071-61-3 876071-62-4 876071-64-6
  876071-66-8 876071-68-0 876071-71-5 876071-73-7 876071-75-9
  876071-77-1 876071-79-3 876071-81-7 876071-83-9 876072-34-3
  876072-36-5 876072-38-7 876072-40-1 876072-42-3 876072-44-5
  876072-46-7 876072-48-9 876072-50-3 876072-52-5 876072-54-7
  876072-56-9 876072-58-1 876072-61-6 876072-63-8 876072-65-0
  876072-67-2 876072-69-4 876072-71-8 876072-73-0 876072-75-2
  876072-77-4 876072-79-6 876072-81-0 876072-83-2 876072-85-4
  876072-87-6 876072-89-8 876072-91-2 876072-93-4 876072-95-6
  876072-97-8 876073-00-6 876073-02-8 876073-04-0 876073-16-4
  876073-18-6 876073-20-0 876073-30-2 876073-29-9 876073-24-4
  876073-25-5 876073-26-6 876073-27-7 876073-28-8 876073-21-1
  876073-22-2 876073-23-3 nucleotide sequence; methods of diagnosing
  and treating obesity, diabetes and insulin resistance by modulators of
  biomarkers identified by gene expression profiling in adipose tissues
306748-07-2 sequence homolog, gene DUSP4; methods of diagnosing and
  treating obesity, diabetes and insulin resistance by modulators of
  biomarkers identified by gene expression profiling in adipose tissues
9031-96-3 sequence homolog, gene SCRN2, for secernin 2; methods of
  diagnosing and treating obesity, diabetes and insulin resistance by
  modulators of biomarkers identified by gene expression profiling in
  adipose tissues

2/7/26   (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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143151735   CA: 143(9)151735r   DISSERTATION
Identification and characterization of a novel leukocyte chemoattractant
and receptor
AUTHOR(S): Zabel, Brian Anthony
LOCATION: Stanford Univ., Stanford, CA, USA
DATE: 2004 PAGES: 103 pp. CODEN: DABBBA LANGUAGE: English CITATION:
Diss. Abstr. Int., B 2004, 65(4), 1640 AVAIL: UMI, Order No. DA3128504
  SECTION:
  CA215010 Immunochemistry
IDENTIFIERS: chemoattractant receptor CMKLR1 TIG2 protein leukocyte,
  plasmacytoid dendritic cell chemoattractant receptor human
DESCRIPTORS:
Chemokine receptors...
  CMKLR1 (chemokine-like receptor 1); human leukocyte chemoattractant
  receptor CMKLR1 and its TIG2 ligand
Human...
  human leukocyte chemoattractant receptor CMKLR1 and its TIG2 ligand
Dendritic cell...
  plasmacytoid; human leukocyte chemoattractant receptor CMKLR1 and its
  TIG2 ligand
Proteins...
  TIG2; human leukocyte chemoattractant receptor CMKLR1 and its TIG2
  ligand

2/7/27   (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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140158535   CA: 140(11)158535x   PATENT
Gene expression profiling of Gleason grades 3 and 4/5 prostate cancer for
identifying tumor markers, and diagnostic and therapeutic uses
INVENTOR(AUTHOR): Mahadevappa, Mamatha; Zhang, Zhaomei; Warrington, Janet
A.; Palma, John F.; Caldwell, Mitchell C.; Chen, Zuxiong; Fan, Zhenbin;
Mcneal, John E.; Nolley, Rosalie; Stamey, Thomas A.
LOCATION: USA
ASSIGNEE: Affymetrix, Inc.
PATENT: U.S. Pat. Appl. Publ. ; US 20040029151 A1 DATE: 20040212
APPLICATION: US 411537 (20030409) *US PV371304 (20020409)
PAGES: 40 pp. CODEN: USXXCO LANGUAGE: English
PATENT CLASSIFICATIONS:
  CLASS: 435006000; C12Q-001/68A
  SECTION:
  CA203001 Biochemical Genetics
  CA201XXX Pharmacology
  CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: mRNA prostate cancer gene expression profile diagnosis
  antitumor screening, neoplasm prostate tumor marker diagnosis
  monitoring, hepsin prostate tumor marker diagnosis monitoring
DESCRIPTORS:
DNA microarray technology...
  Affymetrix GeneChip; gene expression profiling of Gleason grades 3 and
  4/5 prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Antibodies and Immunoglobulins...
  against proteins modulated in prostate cancer; gene expression
  profiling of Gleason grades 3 and 4/5 prostate cancer for identifying
  tumor markers, and diagnostic and therapeutic uses
Gene,animal... Proteins...
  Bcl-2; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Prostate gland,disease...
  benign hyperplasia, control; gene expression profiling of Gleason
  grades 3 and 4/5 prostate cancer for identifying tumor markers, and
  diagnostic and therapeutic uses
Tubulins...
  .beta.-; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Potassium channel...
  beta subunit; gene expression profiling of Gleason grades 3 and 4/5
  prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Transforming growth factors...
  .beta.3-, versicans; gene expression profiling of Gleason grades 3 and
  4/5 prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Laminins...
  B1; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Diagnosis...
  cancer; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Prostate gland,neoplasm...
  carcinoma; gene expression profiling of Gleason grades 3 and 4/5
  prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Antigens...
  carcinoma-assocd., GA733-2; gene expression profiling of Gleason grades
  3 and 4/5 prostate cancer for identifying tumor markers, and diagnostic

and therapeutic uses
Proteins...
  cardiac gap junction; gene expression profiling of Gleason grades 3 and
  4/5 prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Proteins...
  claudins, 8; gene expression profiling of Gleason grades 3 and 4/5
  prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Transcription factors...
  ETR101; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Osteonectin... Desmins... Prostate gland,neoplasm... Gene expression
profiles,animal... Tumor markers... Drug targets... Drug screening... Gene
therapy... Human... Prognosis... Glucocorticoid receptors... Fibroblast
growth factor receptors... Antisense oligonucleotides... Syndecans...
Estrogen receptors... Decorins... Thrombospondins...
  gene expression profiling of Gleason grades 3 and 4/5 prostate cancer
  for identifying tumor markers, and diagnostic and therapeutic uses
Diagnosis...
  genetic; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Proteins...
  glutaredoxins; gene expression profiling of Gleason grades 3 and 4/5
  prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Proteins...
  gravin; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Proteins...
  hevin, -like protein; gene expression profiling of Gleason grades 3 and
  4/5 prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Transcription factors...
  ID4 (inhibitor of differentiation 4), Id-related helix-loop-helix
  protein; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and thera
Insulin-like growth factor-binding proteins...
  IGFBP-6; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Annexins...
  II; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Proteins...
  LACI (lipoprotein-assocd. coagulation inhibitor); gene expression
  profiling of Gleason grades 3 and 4/5 prostate cancer for identifying
  tumor markers, and diagnostic and therapeutic uses
Proteins...
  LIM domain-contg., smooth muscle; gene expression profiling of Gleason
  grades 3 and 4/5 prostate cancer for identifying tumor markers, and
  diagnostic and therapeutic uses
Sodium channel...
  lung amiloride sensitive; gene expression profiling of Gleason grades 3
  and 4/5 prostate cancer for identifying tumor markers, and diagnostic
  and therapeutic uses
Proteins...
  macrophage capping; gene expression profiling of Gleason grades 3 and
  4/5 prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Proteins...
  matrilin, 2; gene expression profiling of Gleason grades 3 and 4/5
  prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Diagnosis...
  mol.; gene expression profiling of Gleason grades 3 and 4/5 prostate

cancer for identifying tumor markers, and diagnostic and therapeutic
uses
Antigens...
  M6; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Proteins...
  nel-related protein 2; gene expression profiling of Gleason grades 3
  and 4/5 prostate cancer for identifying tumor markers, and diagnostic
  and therapeutic uses
Proteins...
  NELL2 (nel (chicken-like) 2); gene expression profiling of Gleason
  grades 3 and 4/5 prostate cancer for identifying tumor markers, and
  diagnostic and therapeutic uses
Proteins...
  neuronal membrane glycoprotein M6b; gene expression profiling of
  Gleason grades 3 and 4/5 prostate cancer for identifying tumor markers,
  and diagnostic and therapeutic uses
Proteins...
  nucleotide-binding; gene expression profiling of Gleason grades 3 and
  4/5 prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
mRNA...
  of differentially expressed genes; gene expression profiling of Gleason
  grades 3 and 4/5 prostate cancer for identifying tumor markers, and
  diagnostic and therapeutic uses
G protein-coupled receptors...
  orphan; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Myosins...
  Ovarian cancer down-regulated myosin heavy chain homolog (Doc1); gene
  expression profiling of Gleason grades 3 and 4/5 prostate cancer for
  identifying tumor markers, and diagnostic and therapeutic use
Proteins...
  procollagen C-proteinase enhancer; gene expression profiling of Gleason
  grades 3 and 4/5 prostate cancer for identifying tumor markers, and
  diagnostic and therapeutic uses
Collagens,biological studies...
  procollagens, prepro-alpha2(I); gene expression profiling of Gleason
  grades 3 and 4/5 prostate cancer for identifying tumor markers, and
  diagnostic and therapeutic uses
Proteins...
  RACH1; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Proteins...
  retinol-binding, cellular; gene expression profiling of Gleason grades
  3 and 4/5 prostate cancer for identifying tumor markers, and diagnostic
  and therapeutic uses
Antitumor agents... Carcinogens...
  screening; gene expression profiling of Gleason grades 3 and 4/5
  prostate cancer for identifying tumor markers, and diagnostic and
  therapeutic uses
Chemokines...
  SDF-1 (stromal-derived factor-1), versicans; gene expression profiling
  of Gleason grades 3 and 4/5 prostate cancer for identifying tumor
  markers, and diagnostic and therapeutic uses
Proteins...
  secretory, 57, prostatic; gene expression profiling of Gleason grades 3
  and 4/5 prostate cancer for identifying tumor markers, and diagnostic
  and therapeutic uses
Transcription factors...
  SNF2a; gene expression profiling of Gleason grades 3 and 4/5 prostate
  cancer for identifying tumor markers, and diagnostic and therapeutic
  uses
Initiation factors(protein formation)...
  Tif (translation initiation factor); gene expression profiling of
  Gleason grades 3 and 4/5 prostate cancer for identifying tumor markers,
  and diagnostic and therapeutic uses

Proteins...
   TIG2 (tazarotene-induced 2); gene expression profiling of Gleason
   grades 3 and 4/5 prostate cancer for identifying tumor markers, and
   diagnostic and therapeutic uses
Proteins...
   TRF1 (telomeric repeat binding factor); gene expression profiling of
   Gleason grades 3 and 4/5 prostate cancer for identifying tumor markers,
   and diagnostic and therapeutic uses
Endothelin receptors...
   type ETA; gene expression profiling of Gleason grades 3 and 4/5
   prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
Proteoglycans,biological studies...
   versicans, splice-variant precursor peptide; gene expression profiling
   of Gleason grades 3 and 4/5 prostate cancer for identifying tumor
   markers, and diagnostic and therapeutic uses
Proteins...
   zinc finger-contg., 185; gene expression profiling of Gleason grades 3
   and 4/5 prostate cancer for identifying tumor markers, and diagnostic
   and therapeutic uses
Transport proteins...
   zinc-transporting; gene expression profiling of Gleason grades 3 and
   4/5 prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
Keratins...
   15; gene expression profiling of Gleason grades 3 and 4/5 prostate
   cancer for identifying tumor markers, and diagnostic and therapeutic
   uses
Keratins...
   5; gene expression profiling of Gleason grades 3 and 4/5 prostate
   cancer for identifying tumor markers, and diagnostic and therapeutic
   uses
 CAS REGISTRY NUMBERS:
9029-62-3 adult male liver; gene expression profiling of Gleason grades 3
   and 4/5 prostate cancer for identifying tumor markers, and diagnostic
   and therapeutic uses
444951-09-1 459581-31-8 459582-49-1 479330-79-5 479478-16-5
   480121-54-8 481151-71-7 481208-24-6 481228-13-1 445046-92-4
   129817-71-6 156288-53-8 193160-07-5 252907-41-8 252907-42-9
   391964-32-2 391966-02-2 391966-19-1 391972-62-6 391975-12-5
   431954-28-8 444968-26-7 445046-96-8 445047-17-6 459511-41-2
   459517-32-9 459518-75-3 459531-92-1 459536-10-8 459536-88-0
   459543-02-3 459543-08-9 459580-61-1 459594-39-9 459597-19-4
   459752-16-0 462261-47-8 475229-35-7 479330-43-3 479330-77-3
   479478-61-0 480122-43-8 480128-51-6 480287-67-0 480651-21-6
   480688-69-5 480937-27-7 481140-37-8 481144-98-3 481147-78-8
   481149-96-6 481175-92-2 481177-17-7 481195-83-9 481239-76-3
   481240-69-1 481245-38-9 481283-50-5 481287-30-3 481287-38-1
   481330-89-6 amino acid sequence; gene expression profiling of Gleason
   grades 3 and 4/5 prostate cancer for identifying tumor markers, and
   diagnostic and therapeutic uses
9001-15-4 b; gene expression profiling of Gleason grades 3 and 4/5
   prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
61512-21-8 .beta.; gene expression profiling of Gleason grades 3 and 4/5
   prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
330596-22-0 dioxin-inducible; gene expression profiling of Gleason grades
   3 and 4/5 prostate cancer for identifying tumor markers, and diagnostic
   and therapeutic uses
9015-81-0 80146-85-6 9027-35-4 9029-07-6 9036-20-8 60748-73-4
   197980-93-1 65802-85-9 97501-93-4 63551-76-8 51845-53-5 9001-60-9
   37277-82-0 9045-77-6 82062-90-6 9032-04-6 9011-97-6 107544-29-6
   37213-56-2 80295-57-4 9035-74-9 157857-10-8 gene expression
   profiling of Gleason grades 3 and 4/5 prostate cancer for identifying
   tumor markers, and diagnostic and therapeutic uses
9061-61-4 HBNF-1; gene expression profiling of Gleason grades 3 and 4/5
   prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
112398-23-9 highly upregulated antigen in prostate cancer, therapeutic

antibody to; gene expression profiling of Gleason grades 3 and 4/5
   prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
9059-32-9 homolog HeLA cell line, 833 nt; gene expression profiling of
   Gleason grades 3 and 4/5 prostate cancer for identifying tumor markers,
   and diagnostic and therapeutic uses
9031-72-5 I, alpha subunit; gene expression profiling of Gleason grades 3
   and 4/5 prostate cancer for identifying tumor markers, and diagnostic
   and therapeutic uses
9031-86-1 kidney 1435 nt; gene expression profiling of Gleason grades 3
   and 4/5 prostate cancer for identifying tumor markers, and diagnostic
   and therapeutic uses
54249-88-6 liver; gene expression profiling of Gleason grades 3 and 4/5
   prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
137367-20-5 NADP dependent; gene expression profiling of Gleason grades 3
   and 4/5 prostate cancer for identifying tumor markers, and diagnostic
   and therapeutic uses
139847-59-9 140030-63-3 140034-78-2 140279-66-9 140285-75-2
   140333-60-4 144531-48-6 148450-54-8 156797-75-0 164843-43-0
   165472-96-8 165706-55-8 167246-69-7 172177-78-5 172866-06-7
   173661-84-2 175113-26-5 175384-95-9 176455-69-9 176800-07-0
   178659-11-5 184566-24-3 185243-06-5 186637-50-3 188379-53-5
   384422-15-5 384479-92-9 384493-08-7 384508-17-2 384519-36-2
   384543-55-9 384602-52-2 384653-03-6 384681-98-5 384737-67-1
   385090-26-6 389180-49-8 389182-33-6 389182-71-2 389204-63-1
   389210-89-3 391527-17-6 391528-22-6 391528-63-5 391531-30-9
   391532-55-1 391536-31-5 391536-66-6 391537-46-5 391537-52-3
   391539-53-0 391540-04-8 391546-98-8 391548-05-3 391548-81-5
   391550-44-0 391560-56-8 391563-77-2 391763-33-0 391763-54-5
   391775-83-0 391826-32-7 391831-84-8 392215-85-9 398425-00-8
   384431-51-0 389182-31-4 389185-03-9 391530-07-7 391765-30-3
   392207-45-3 nucleotide sequence; gene expression profiling of Gleason
   grades 3 and 4/5 prostate cancer for identifying tumor markers, and
   diagnostic and therapeutic uses
50812-37-8 .pi., .pi.; gene expression profiling of Gleason grades 3 and
   4/5 prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
9001-84-7 PLA2G7/LDL-; gene expression profiling of Gleason grades 3 and
   4/5 prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
96282-35-8 SERPINF1; gene expression profiling of Gleason grades 3 and 4/5
   prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
9001-16-5 subunit VIIa muscle isoforms; gene expression profiling of
   Gleason grades 3 and 4/5 prostate cancer for identifying tumor markers,
   and diagnostic and therapeutic uses
64885-96-7 subunit 58; gene expression profiling of Gleason grades 3 and
   4/5 prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses
67763-97-7 145809-21-8 148348-15-6 157857-21-1 186270-49-5 versicans;
   gene expression profiling of Gleason grades 3 and 4/5 prostate cancer
   for identifying tumor markers, and diagnostic and therapeutic uses
9001-99-4 4; gene expression profiling of Gleason grades 3 and 4/5
   prostate cancer for identifying tumor markers, and diagnostic and
   therapeutic uses

2/7/28   (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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138131057 CA: 138(10)131057q PATENT
Identification and interaction of TIG2 (Tazarotene-Induced Gene-2)
polypeptides with natural ligand of G-protein coupled receptor Chemr23
and uses thereof
INVENTOR(AUTHOR): Wittamer, Valerie; Communi, David; Vandenbogaerde, Ann;
Detheux, Michel; Parmentier, Marc
LOCATION: Belg.
ASSIGNEE: Euroscreen SA

PATENT: PCT International ; WO 200306996 A2 DATE: 20030123
APPLICATION: WO 2002EP7647 (20020709) *US PV303858 (20010709) *US 905253 (20010713)
PAGES: 99 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: G01N-033/74A; G01N-033/566B; A61K-038/00B; C12Q-001/68B; C07K-016/00B; C07K-014/00B
DESIGNATED COUNTRIES: CA; JP DESIGNATED REGIONAL: AT; BE; BG; CH; CY; CZ ; DE; DK; EE; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; SK; TR
SECTION:
CA201001 Pharmacology
IDENTIFIERS: TIG2 gene protein polypeptide chemoattractant receptor ChemR23 disease diagnosis
DESCRIPTORS:
Protein sequences...
alignment; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Artery...
aorta, expression in; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Infection...
bacterial; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Aequorins...
based assay; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Transplant and Transplantation...
bone; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Gene,animal...
Chemr23 mutation; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
G protein-coupled receptors...
ChemR23; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Intestine...
colon, expression in; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Brain... Bone marrow... Spleen... Lymph node... Thymus gland... Stomach... Pancreas... Liver... Kidney... Testis... Ovary... Placenta... Pituitary gland... Thyroid gland... Adrenal gland... Lung... Trachea(anatomical)... Heart... Muscle... Skin... Adipose tissue... Monocyte... T cell(lymphocyte) ... Dendritic cell...
expression in; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Reporter gene...
expression; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Fertility...
female, disorder; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Disease,animal...
genetic; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Proteins...
green fluorescent; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof

Bone...
healing; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Phosphatidylinositols... Diglycerides... Drug screening... Diagnosis... Test kits... Human... Protein sequences... cDNA sequences... Radionuclides,biological studies... Fluorescent substances... Fluorescence ... Epitopes... Surface plasmon resonance... Polarized fluorescence... Fluorescence quenching... Energy transfer... Antibodies... Molecular cloning... Signal transduction,biological... Nucleic acids... mRNA... Thioredoxins... Antitumor agents... Neoplasm... Anti-inflammatory agents... Inflammation... Autoimmune disease... AIDS(disease)... Osteoporosis... Transplant rejection... Psoriasis... Eczema... Skin,disease... Antiviral agents... Antibacterial agents... Parasiticides... Ovary,neoplasm... Uterus,neoplasm... Microarray technology...
identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Neoplasm...
metastasis; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Intestine...
small, expression in; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Liposomes...
synthetic; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Proteins...
TIG2 (tazarotene-induced gene 2); identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Gene,animal... Gene,animal... Fusion proteins(chimeric proteins)...
TIG2; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Bone...
transplant; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Infection...
viral; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
Gene,animal...
2; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
CAS REGISTRY NUMBERS:
491666-74-1 491666-76-3 491666-89-8 491666-78-5 491666-79-6 491666-80-9 491666-81-0 491666-82-1 491666-83-2 491666-84-3 491666-85-4 491666-86-5 491666-87-6 491666-88-7 amino acid sequence; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
69-79-4 binding protein; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
7440-70-2 biological studies, identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
71-00-1 biological studies, tags; identification and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of G-protein coupled receptor Chemr23 and uses thereof
9012-42-4 60-92-4 141436-78-4 506-30-9 142243-02-5 80449-02-1 27121-73-9 50812-37-8 9001-78-9 283131-95-3 207788-36-1 186637-50-3 337100-43-3 297087-93-5 337100-01-3 337099-55-5 254579-96-9 268395-62-6 297065-29-3 312851-10-8 336036-14-7

identification and interaction of TIG2 (Tazarotene-Induced Gene-2)
polypeptides with natural ligand of G-protein coupled receptor Chemr23
and uses thereof
491666-75-2 491666-77-4 491666-90-1 nucleotide sequence; identification
and interaction of TIG2 (Tazarotene-Induced Gene-2) polypeptides with
natural ligand of G-protein coupled receptor Chemr23 and uses thereof
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unclaimed nucleotide sequence; identification and interaction of TIG2
(Tazarotene-Induced Gene-2) polypeptides with natural ligand of
G-protein coupled receptor Chemr23 and uses thereof
491667-82-4 unclaimed protein sequence; identification and interaction of
TIG2 (Tazarotene-Induced Gene-2) polypeptides with natural ligand of
G-protein coupled receptor Chemr23 and uses thereof
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491667-80-2 491667-81-3 491667-93-7 491667-94-8 491667-95-9
491667-96-0 491667-97-1 unclaimed sequence; identification and
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